

Is the antisocial child father of the abusive man? A 40-year prospective longitudinal study on the developmental antecedents of intimate partner violence<sup>1</sup>

Patrick Lussier<sup>a</sup>, David P. Farrington<sup>b</sup> & Terrie E. Moffitt<sup>c</sup>

<sup>a</sup>Assistant professor, School of Criminology at Simon Fraser University  
Codirector of the Center for Research on Sexual Violence  
Member of the Center for Social Responsibility at Simon Fraser University  
Coprincipal investigator of the Vancouver Longitudinal Study

<sup>b</sup>Professor of psychological criminology at the Institute of Criminology, Cambridge University  
Adjunct professor of psychiatry at Western Psychiatric Institute and Clinic, University of Pittsburgh

<sup>c</sup>Institute of Psychiatry, King's College, London

### Abstract

This prospective longitudinal study examined whether early childhood risk factors contributed to explaining and predicting intimate partner violence (IPV) in mid-adulthood. Participants included 202 men from the Cambridge longitudinal study who were in an intimate relationship in their mid-40s. Neuropsychological deficits and the presence of a criminogenic family environment were measured between ages 8 and 10. Antisocial behavior was measured between ages 8 and 18. IPV was measured at age 48 using a self-report instrument completed by the participants' female partners. Perpetration and victimization rates were relatively high; violence was mostly mutual, and men were more likely to be victims than perpetrators. Findings indicate that a criminogenic environment increases the risk of IPV by fostering the development of antisocial behavior and neuropsychological deficits. A link also exists between a high level of antisocial behavior during adolescence and the risk of IPV later in life. The results suggest the presence of both continuity and discontinuity of antisocial behavior as childhood risk factors that increase the likelihood of future involvement in IPV, but the role of these risk factors is modest.

Keywords: intimate partner violence, longitudinal study, antisocial behavior, early onset, neuropsychological deficits, intergenerational transmission, life-course persistence

REFERENCE: Lussier, P, Farrington, D. P. & Moffitt, T. E. (2009). Is the antisocial child father of the abusive man? A 40-year prospective longitudinal study on the developmental antecedents of intimate partner violence. *Criminology*, 47 (3).

---

<sup>1</sup> Correspondence concerning this article should be addressed to P. Lussier, Pavillon Charles-De Koninck, 1030, ave. des Sciences-Humaines, Quebec, Quebec G1V 0A6, Canada. Tel.: +1 418 656 2131x5978. E-mail addresses: patrick.lussier@svs.ulaval.ca.

Is the antisocial child father of the abusive man? A 40-years prospective longitudinal study on the developmental antecedents of intimate partner violence

Domestic violence has traditionally been portrayed as male-perpetrated violence against women (also known as patriarchal terrorism, wife beating, and spousal assault) (Dobash et al., 1992; Johnson, 1995). This portrayal emerged from empirical studies based mainly on clinical samples of severe victimization cases. In the United States, court-mandated batterer treatment programs for male perpetrators have been the most widely used method for dealing with domestic violence. However, this policy and its associated guidelines have not been well supported by empirical evidence. Recent quantitative reviews indicated that batterer treatment programs for violent male partners had minimal impact (Babcock, Green, and Robie, 2004; Feder and Wilson, 2005). Some researchers argued that the lack of a significant “treatment effect” might be attributable to methodological shortcomings of evaluative studies as well as to variations in the quality of research designs found in the scientific literature (Gondolf, 2004). These poor outcomes fueled a corpus of research that focused on the development and implementation of preventive efforts before the onset of violence (Moffitt and Caspi, 2003; Smithey and Strauss, 2004; Whitaker et al., 2006). This expansion of interventions toward primary prevention programs was accompanied by a reconsideration of the scope of domestic violence. The portrayal of domestic violence, which is characterized primarily as an act between a male perpetrator and a female victim, was challenged and expanded after the results of large surveys based on community samples. Those studies showed that violence was experienced by both men and women, was mostly reciprocal between partners, was not limited to physical violence and included verbal aggression, and was not confined to married couples (it also occurred in dating and cohabiting couples) (Archer, 2002; Felson, 2002; Magdol et al., 1997; Straus, 1990). Consequently, these findings led researchers to adopt a broader perspective by investigating the

precursors of intimate partner violence (IPV), that is, common couple violence that included both verbal aggression and physical violence. The interest in long-term preventive efforts of IPV, however, has grown faster than empirical knowledge about its significant risk factors. Using a developmental, life-course perspective, recent prospective longitudinal studies suggested that successful interventions for childhood antisocial behavior may have long-term impacts on IPV in adulthood. The current study examines this claim by reviewing the long-term risk factors of IPV in adulthood, to inform policy makers about effective prevention programs.

#### Developmental Approach to IPV

Since the advent of developmental criminology (Loeber and LeBlanc, 1990), various theoretical perspectives have been proposed to improve the understanding of antisocial behavior over the life course (Farrington, 2003, 2005; Lahey and Waldman, 2003; LeBlanc, 2005; Moffitt, 1993; Thornberry, 2005). Developmental theorists argued that it stemmed from an underlying antisocial propensity reflected in a relative rank ordering of individuals and maintained over time (Farrington, 2003; Lahey and Waldman, 2003; Moffitt, 1993). Researchers have not yet established at what point in development this propensity is initiated and maintained. Two important mechanisms were described to explain the propensity: the static (population heterogeneity) and dynamic (state-dependent) processes (Nagin and Paternoster, 2000). Propensity theorists emphasized the role of static (i.e., the long-lasting impact of early individual differences) (e.g., Gottfredson and Hirschi, 1990), whereas developmentalists stressed state-dependent processes (i.e., earlier behavior affects the probability of later behavior) (e.g., Thornberry, 2005). Other developmentalists, however, argued for the joint effect of static and dynamic processes through successive interactions and transactions between a predisposed child and a criminogenic environment (Caspi, 2000; Lahey and Waldman, 2003; Moffitt, 1993). The following three types of child–environment interactions favor the continuity of antisocial

behavior over the life course: (1) inherited early deficits that limit the child's development of self-regulation and self-control abilities; (2) these deficits, in turn, may evoke negative emotional and behavioral reactions from the parents, characterized by similar deficits, which can entrench maladaptive behaviors of the child; and (3) reactions from the social environment may contribute to the child selecting an antisocial social environment (e.g., peers and partners) that reinforce existing maladaptive behavioral patterns. Early maladaptive behaviors may increase the risk of long-term negative outcomes by limiting prosocial activities, which favors the accumulation of personal and social disadvantages (e.g., with respect to employment, income, housing, and intimate relationships) and confines the behavioral repertoire to antisocial actions and reactions (i.e., reckless, defiant, aggressive, and dishonest). Therefore, the earlier these three processes are initiated, the greater the likelihood they will lead to persistent antisocial behavior over the life course.

Developmental models of antisocial behavior tend to agree on the different mechanisms responsible for the development of underlying antisocial propensity. Generally, developmental models recognized that predisposing characteristics of the child and the presence of a criminogenic social environment explained antisocial persistence across the life course. The child's predisposition has generally been defined according to his\her individual biological capacities. More specifically, developmental models recognized the predisposing role of low cognitive abilities, poor problem-solving skills, inattention problems, and impulsivity in the commission of antisocial acts (Farrington, 2003; Moffitt, 1993). Subtle neuropsychological deficits can lead to stable individual differences in cognitive (i.e., attention and verbal reasoning), temperamental (i.e., emotional reactivity), and behavioral processes (i.e., speech, motor coordination, and impulse control), which limit childhood abilities for self-control and self-regulation (Lahey and Waldman, 2003; Moffitt, 1993). However, the criminogenic environment

has generally been described as a composite of poor parenting skills, antisocial modeling, socioeconomic deprivation, and low attachment between the child and the parents (Farrington, 2003; LeBlanc, 2005; Thornberry, 2005). These risk factors have been described as a combination of the following four mechanisms (Farrington, 2003; LeBlanc, 2005; Thornberry, 2005): 1) inadequate constraining of the child's behavior through supervision and sanctions; 2) absence of a child-parent bond; 3) modeling and reinforcing of antisocial behavior (as opposed to prosocial behavior); and 4) socioeconomic deprivation, which puts additional stress and pressure on the family and limits the accumulation of human and social capital. Although developmental models of antisocial behavior recognized both the child's predisposing characteristics and the criminogenic environment, models differed with respect to their emphasis. For example, some theories emphasized individual differences (Lahey and Waldman, 2003), whereas others emphasized the role of the criminogenic environment (LeBlanc, 2005).

#### Criminogenic Family Environment and IPV

Researchers who examined the developmental antecedents of IPV have been concerned with only one aspect of the criminogenic environment, that is, early inadequate modeling. Based on retrospective data on interparental violence, these researchers reported a small but significant main effect, with a stronger effect for clinical samples as opposed to community samples of perpetrators (Schumacher et al., 2001; Stith et al., 2004). In recent years, some researchers have looked prospectively and longitudinally at the impact of the modeling of violence on later IPV perpetration and victimization. The findings from these empirical studies did not provide strong evidence for a simple imitation hypothesis; rather, they indicated that the strength of the association between witnessing interparental violence and later IPV was generally small and nonsignificant (Farrington, 1994; Lavoie et al., 2002; Magdol et al., 1998; Simons, Lin, and Gordon, 1998; see, however, Ehrensaft et al., 2003). These findings may be attributable to the

presence of more complex developmental processes at play. Other characteristics of a criminogenic family environment are linked to later IPV, such as parents' low income, unemployment, criminal behavior, and poor parenting skills (Capaldi and Clark, 1998; Farrington, 1994; Lavoie et al., 2002; Magdol et al., 1998; Simons, Lin, and Gordon, 1998; Woodward, Fergusson, and Horwood, 2002). The presence of a multi-risk criminogenic family environment seems more important than any one specific family risk factor. For example, on the one hand, the link between exposure to interparental violence and later IPV disappeared after controlling for characteristics of a criminogenic environment (e.g., childhood sexual abuse, maternal age, low standard of living, and poor maternal care) (Fergusson, Boden, and Horwood, 2006). On the other hand, the impact of an early criminogenic family environment on later involvement in IPV is mediated by the development of antisocial behavior and the presence of antisocial personality traits (Lavoie et al., 2002; Simons, Lin, and Gordon, 1998; White and Widom, 2003).

#### Neuropsychological Deficits and IPV

Researchers have shown that individual differences play a role in IPV perpetration and victimization, but they have generally neglected the possible role of neuropsychological deficits (Fagan and Browne, 1994; Holtzworth-Munroe et al., 1997; Moffitt et al., 2000; Schumacher et al., 2001; Stith et al., 2004). It can be hypothesized from developmental models of antisocial behavior that neuropsychological deficits can lead to IPV through the development of an antisocial propensity (Farrington, 2003; Lahey and Waldman, 2003; Moffitt, 1993). Preliminary findings from clinical samples of male batterers suggested that they are more likely to be characterized by neuropsychological deficits than controls (Cohen et al., 2003). Empirical studies based on a community sample have overlooked the possible role of neuropsychological deficits

on the perpetration of IPV<sup>2</sup>. These deficits have been difficult to screen and assess, which might partly explain the lack of related empirical studies. To our knowledge, only three prospective longitudinal studies have examined the relationship between early neuropsychological deficits and involvement in partner violence in adulthood (Farrington, 1994; Magdol et al., 1998; Woodward, Fergusson, and Horwood, 2002). These studies yielded mixed results and suggested that these deficits might play a role in the development of a life-course persistent (LCP) antisocial trajectory. The first pattern identified is a direct link between factors, which suggests neuropsychological deficits and IPV. Magdol et al. (1998) reported that low IQ and poor reading abilities were related to IPV perpetration. Those associations held after controlling for characteristics associated with a criminogenic environment (i.e., low socioeconomic resources and poor family relationship). The second pattern identified is the lack of association between measures of neuropsychological deficits and later IPV. For example, Farrington (1994) did not find a link between low verbal IQ and attention deficits measured at ages 8–10 and wife beating assessed at age 32. Finally, an indirect effect might be at play. Woodward, Fergusson, and Horwood (2002) found that measures of IQ and attention deficits in childhood were both related to LCP antisocial trajectory, which in turn was strongly related to IPV perpetration and victimization at age 21. It is difficult to draw firm conclusions from those findings with respect to the possible role of neuropsychological deficits on later IPV.

#### Childhood Antisocial Behavior and IPV

The continuity between antisocial behavior and IPV seems to be stronger for those who show an early onset of antisocial behavior. Moffitt and Caspi (2003) recently argued that

---

<sup>2</sup> Existing studies have mainly examined cognitions supporting violence and men's domination over women, rather than stable cognitive abilities. Findings from qualitative (Holtzworth-Munroe et al., 1997; Schumacher et al., 2001) and quantitative (Stith et al., 2004) reviews suggest that male perpetrators of IPV are more likely to support attitudes promoting male domination and interpersonal violence. It is still unknown whether cognitions that support domination and interpersonal violence are related to cognitive abilities.

problematic childhood behavior was the strongest developmental risk factor for partner violence. Individuals with a history of defiance, aggression, recklessness, dishonesty, and deceit were likely to show those behaviors with their intimate adult partner, creating situations that might escalate to IPV. Several empirical investigations based on prospective longitudinal data supported the hypothesis of the continuity between childhood antisocial behavior and adult IPV<sup>3</sup>.

First, individuals with an early onset of antisocial behavior were more likely to show evidence of later partner violence than individuals with an adolescent onset. For example, in the Christchurch Study, 36 percent of early onset individuals had committed acts of physical violence against a partner by age 21, compared with 19 percent for late onset individuals and 10 percent for the nonantisocial group (Woodward, Fergusson, and Horwood, 2002). Second, individuals characterized by a childhood onset have been shown to be responsible for a disproportionate percentage of all acts of violence against women. In the Dunedin study, males following the LCP trajectory, which represents 10 percent of the birth cohort, were responsible for 62 percent of all

---

<sup>3</sup> The concept of heterotypic continuity was an interesting starting point in the link between early antisocial behavior and IPV. Early developmental studies revealed a marked continuity of aggression from childhood to adulthood (Farrington, 1994; Huesmann and Eron, 1992), but these studies might have produced an incomplete picture of continuity. Heterotypic continuity was important because it linked different conceptually related antisocial manifestations (i.e., heterotypic) across different developmental periods (i.e., continuity), which is explained by a single underlying latent trait (antisocial propensity). From a developmental perspective, heterotypic continuity was crucial because it conceptualized IPV as another antisocial manifestation. Heterotypic continuity suggested that different kinds of antisocial behaviors were committed at different ages in an orderly and predictable sequence. Antisocial behavior, if it persists, was therefore described as a moving target to which new behavioral manifestations were added over new developmental periods (LeBlanc, 2005; Loeber and LeBlanc, 1990; Moffitt, 1993; Patterson, 1993). Based on heterotypic continuity, antisocial behavior was best perceived as a series of developmental stages in which behavioral manifestations displayed both continuity and change. During early childhood, antisocial behavior takes the form of authority conflict behavior at home (e.g., defiance) and overt behavior (e.g., physical aggression). In later childhood, this resistance to authority is manifested in school (e.g., truancy), and covert behaviors are added (frequent lying, shoplifting, etc.). In adolescence, new forms of overt (e.g., gang fighting) and covert (serious theft and breaking and entering) behaviors emerge, with the introduction of reckless behavior (e.g., substance use and risky, promiscuous sexual activity). In adulthood, new behaviors are added, such as verbal and physical violence against an intimate partner. This hypothesis is consistent with previous observations that indicated 1) the lack of criminal specialization of partner abusers, 2) the co-occurrence of IPV and other antisocial behavior, 3) the similarities in risk factors for both antisocial behavior and IPV, and 4) the continuity between early antisocial behavior in youth and later IPV (Fagan and Browne, 1994; Moffitt et al., 2000; Moffitt and Caspi, 2003). The concept of heterotypic continuity suggests that one underlying propensity might be responsible for all those behavioral manifestations, which change as new social opportunities present themselves throughout the life course.



convictions of violence against women for that cohort up to age 26 (Moffitt et al., 2002). Third, multivariate statistical analyses have shown the importance of childhood antisocial behavior as a risk factor for partner violence perpetration and victimization, even after controlling for other developmental child and family risk factors. Farrington (1994) reported that the best predictors of IPV at age 32 (which was defined as wife beating) were measures of daring at ages 8–10, being unpopular, having a convicted parent, and having separated parents. After controlling for early childhood environmental risk factors (i.e., social class, family conflict, and harsh physical discipline), Magdol et al. (1998) found that a history of antisocial behavior at ages 7–9 predicted perpetration and victimization of partner violence at age 21. The results held for both men and women. Similarly, Woodward, Fergusson, and Horwood (2002) reported that after controlling for gender, parental physical punishment, interparental violence, attention deficits, an early onset of antisocial behavior predicted both IPV perpetration and victimization. Fourth, both Magdol et al. (1998) and Ehrensaft et al. (2003) reported that evidence of conduct disorder was the best predictor of partner violence perpetration and victimization in early adulthood. Together, these results provided evidence for persistent population heterogeneity but not necessarily a state-dependence effect on the link between antisociality and partner violence.

#### Aim of the study

According to developmental models, the impact of early individual differences is pervasive and can have important ramifications for life-course development, which includes the likelihood of being involved in an aggressive and physical violent intimate relationship in adulthood. Most empirical studies that examine the developmental antecedents of IPV are based on highly selected groups of men referred for intervention by the courts or clinical samples of violent men. The research design of the current study uses a combination of four features rarely observed in most previous investigations, as follows: 1) a prospective longitudinal design; 2) a

representative sample of male abusers; 3) a long-term follow-up period covering childhood, adolescence, and adulthood; and 4) an outcome variable (IPV) based on the partner's self-report. More recently, prospective longitudinal studies have addressed those methodological limitations (e.g., Ehrensaft et al., 2003; Magdol et al., 1998; White and Widom, 2003; Woodward, Fergusson, and Horwood, 2002), but many unresolved issues remain. In fact, only a limited number of prospective longitudinal studies have been conducted, and as a result, only a limited set of risk factors has been tested. Using data from an ongoing prospective longitudinal study, we address previous methodological and conceptual limitations by examining the developmental antecedents of IPV.

First, considering that previous longitudinal studies only examined IPV early in adulthood (20–35 years old), little is known about the nature and prevalence of verbal and physical aggression against an intimate partner in middle adulthood. Furthermore, little is known about the early precursors of involvement in IPV beyond the period of early adulthood. From a developmental perspective, investigating precursors beyond the period of early adulthood is interesting, as different risk factors may be responsible for IPV in middle adulthood. The current study provides baseline data on IPV perpetration and victimization, as well as the associated risk factors using a community sample of men followed over four decades. As a result, this study challenges recent claims from life-course theorists (Sampson and Laub, 2005): First, an accurate long-term prediction of antisocial behavior cannot be made, and second, there is too much heterogeneity in life outcomes to consider the deterministic role of early childhood risk factors.

Second, previous empirical studies stressed that antisocial behavior in youth was one of the most significant predictors of IPV perpetration and victimization (Ehrensaft et al., 2003; Magdol et al., 1998; Moffitt and Caspi, 2003). More specifically, it was argued that the earlier antisocial behavior begins, the more likely it will persist in adulthood. The link between

antisocial behavior in youth and IPV has been shown, but it remains to be determined whether childhood represents the best developmental period to maximize the effectiveness of preventive programs aimed at IPV. The current study examines the link among antisocial behavior in three different developmental periods in youth (childhood, early adolescence, and late adolescence) and later involvement IPV in mid-life. Therefore, we assess whether childhood onset is more strongly related to later IPV than adolescent onset.

Third, developmental models of antisocial behavior have stressed the importance of the criminogenic family environment and neuropsychological deficits of the child. Previous longitudinal studies have focused mainly on testing the imitation hypothesis, and only a few examined the role of neuropsychological deficits on later IPV; these studies produced mixed results. Here, the contributing role of early neuropsychological deficits (poor verbal skills and low verbal IQ) and a criminogenic family environment (i.e., socioeconomic deprivation, poor parenting skills, and antisocial modeling) was examined. More specifically, we hypothesized that both neuropsychological deficits and a criminogenic environment are indirectly linked to IPV through the development of antisocial behavior. These hypotheses have not been tested simultaneously in previous empirical studies. Using a series of autoregressive latent modeling, the direct and indirect impacts of these childhood risk factors were tested, controlling for the development of the antisocial behavior in youth.

## Methodology

### Participants

The study was based on data collected in the Cambridge Study in Delinquent Development, which is a prospective longitudinal survey of 411 males from an inner-city working-class area of London, England (Farrington, 2003; Farrington et al., 2006; West and Farrington, 1973). This sample of boys was first contacted in 1961–1962 when they were about 8

years old. Among them, 399 boys were registered in one of the six primary schools within a 1-mile radius of the research office established at the time. Another 12 boys were selected from a special school for educationally subnormal children to make the sample more representative of boys living in the area. From that moment on, each study male was interviewed at different times over their life course, that is, at about ages 10, 14, 16, 18, 21, 25, 32, and 48. At age 48, of the original 411 participants, 17 (4.1 percent) had died, 5 (1.2 percent) could not be traced, and 24 (5.8 percent) refused to be interviewed. In total, 365 of the 394 still living males (92.6 percent) were re-interviewed. Of those who were re-interviewed, 224 were living with an intimate female partner, most were married (80.8 percent;  $n = 181$ ), engaged (5.4 percent;  $n = 12$ ), or in a serious relationship (11.6 percent;  $n = 26$ ). The current study focused on the developmental history of these 224 men living with an intimate partner at age 48.

*Attrition of participants.* Information on IPV at age 48 was available for 202 of the 224 men (see the section on the measurement of IPV for more details). Therefore, 202 of the original sample of 411 boys were included in the current study. To control for potential bias in our sample, we compared the included sample of men with those not included in the study (i.e., died, could not be traced, refused to be interviewed, not in a significant intimate relationship, or female partner did not complete the questionnaire) on all the independent variables in the study. It could be reasonably argued that the status of the 209 unstudied cases may be attributable to their background and individual characteristics, such as coming from a criminogenic background and being more antisocial. Of the seven early developmental risk factors (i.e., neuropsychological deficits and criminogenic environment measures), none emerged as significantly different between those subjects included and those not included in the analysis. Of the ten measures of antisocial behavior in youth, three emerged as statistically significant. Men included in the study of partner violence were more aggressive in early adolescence [ $t(409) = 2.04$ ,  $p < .01$ , Cohen's  $d$

= .40] and were more reckless in both early [ $t(409) = 2.04, p < .01, \text{Cohen's } d = .20$ ] and late adolescence [ $t(409) = 2.04, p < .01, \text{Cohen's } d = .20$ ]. It is difficult to make sense of these differences based on the information available. Considering that the effect sizes of these significant differences are small for two out of three indicators (Cohen's  $d = .20$ ), and about average for the remainder (Cohen's  $d = .40$ ), potential biases were considered to be somewhat minimal for this article. Performing a logistic regression using these three indicators to predict inclusion or exclusion in the study yielded a model explaining (Cox and Snell R-square) only 2 percent of the variance with only 54.5 percent correctly classified, that is, a little more than 4 percent over what is expected by chance alone.

#### Measures

*Neuropsychological deficits in childhood.* Moffitt (1993) emphasized the role of neuropsychological deficits in explaining LCP antisocial trajectory and, more specifically, verbal and executive functions deficits. The current study focuses on verbal deficits that affect listening and reading skills, problem solving, expressive speech, writing, and memory. Two indicators were used to assess the level of verbal deficits of the participants as a child: verbal reasoning and verbal intelligence. In the Cambridge Study, these two indicators were shown to be related to youth violence and/or adult violence (Farrington, 2005). Verbal reasoning was based on the scores obtained at ages 10–11, on a standardized examination used by the schools in grading children for secondary school selection (Farrington, 2003). Those whose score was in the lowest 25th percentile were coded as having low verbal reasoning (0 = best 75 percent; 1 = worst 25 percent). Low verbal intelligence was measured using the Mill Hill Vocabulary Scale (Raven, 1943) at ages 10–11, which consists of a series of words for which the child was asked to explain the meaning. A higher score on the Mill Hill Vocabulary Scale represents a higher verbal intelligence. The variable was coded so that a high score on the scale would reflect low verbal

intelligence: (0 = high verbal intelligence, that is, scores of 19 or more; 1 = medium-high verbal intelligence, that is, scores of 17 or 18; 2 = medium-low verbal intelligence, that is, scores of 15 or 16; and 3 = low verbal intelligence, that is, scores of 14 and lower).

*Criminogenic family environment.* Five indicators were selected to measure an early criminogenic environment, as follows: 1) low family income, 2) low socioeconomic status (SES) of the family of origin, 3) criminal record of the parents, 4) parental conflict, and 5) inadequate parenting. These five indicators have been empirically demonstrated to be related to youth violence and/or adult violence (Farrington, 2005). Unless otherwise specified, information was collected through interviews conducted by psychiatric social workers when the research participants were 8- and 10-year olds. The indicator of low family income refers to an assessment made by social workers that considered the income, family size, and lifestyle (0 = comfortable; 1 = adequate; 2 = inadequate). Those considered inadequate by social workers were coded as having a low income (i.e., 19 percent of the sample). Low SES of the family of origin refers to the occupation of the father when the participants were age 8. A low SES meant the father was either unskilled or chronically unemployed at the time. The variable was coded as follows: (0 = absence of a low SES and 1 = presence of a low SES). In total, 10.5 percent of the sample was characterized by a low SES. A criminal record of the parents refers to whether the mother and/or the father of the participant had been convicted of a crime before the participant's 10th birthday (0 = parent not convicted and 1 = parent was convicted). For this sample, 29 percent of the boys had a convicted parent. A scale was created to measure parental conflict ( $\alpha = .76$ ). This refers to high disagreement between parents of each participant at the time of the assessment (i.e., chronic tension, disagreements in many fields, raging conflicts, or parents completely estranged) [mean = .39; standard deviation (SD) = .73]. A standardized scale of inadequate parenting refers to the parents' behaviors toward the participant at age 8, which included the following items: 1)

negative attitude of the mother (i.e., overprotective, passive, cruel, and neglectful); 2) negative attitude of the father (i.e., passive, cruel, and neglectful); 3) inadequate discipline of the parents (i.e., erratic, lax, and very strict); 4) lack of vigilance (i.e., careless or neglectful); 5) lack of praising (i.e., no special recognition, privileges, or rewards given when the boy is acting in a good manner); and 6) a lack of interest in the boy's education. A high score on this scale ( $\alpha = .62$ ) indicates poor/inadequate child-rearing practices by the parents during the boy's childhood (mean = .13; SD = 3.59). The items selected and the alpha level observed for the scale of inadequate parenting is in line with other measures of parenting (e.g., Essau, Sasagawa, and Frick, 2006).

*Antisocial behavior.* Measuring antisociality is a challenge for criminologists because behavioral manifestations tend to change from one developmental period to another. To grasp the full spectrum of antisocial behavior, we used the categorization proposed and empirically tested by LeBlanc and Bouthillier (2003). This categorization was used to classify rationally the research participants' behavior into the following four domains: 1) overt behavior (i.e., aggression and violence); 2) covert behavior (i.e., sneaky, deceitful, and dishonest behavior); 3) reckless behavior (i.e., risky and dangerous activities); and 4) authority–conflict behavior (i.e., difficulties respecting rules and authority figures at home or school). Using this categorization, we created ten scales of antisocial behaviors that provide indicators at three points in time and are consistent with related developmental models: 1) childhood consisting of measures taken between ages 8 and 12 based on peer, teacher, or parent ratings; 2) early adolescence consisting of measures taken at age 14, based on self-reports and teacher ratings; and 3) late adolescence, consisting of measures taken at age 18 based on self-reports. In total, ten scales were created, which were composed of multiple items coded as either (1) present or (0) absent. The scores were

summed and then standardized for additional analysis. More details about the coding can be found elsewhere (Farrington, 1994, 2003).

For the dimension of overt behaviors, the following three scales were created: a scale for childhood (2 items;  $\alpha = .73$ , rough at playtime or aggressive at school; mean = 1.91; SD = .68); a scale for early adolescence (8 items;  $\alpha = .66$ ; rough at playtime, quarrelsome, fights outside home, insults strangers, engages in serious fights where people get hurt, carries a weapon, involved in gang fight, or uses a weapon in a fight; mean = 1.53; SD = 1.64); and a scale for late adolescence (4 items,  $\alpha = .80$ ; engages in serious fights where people get hurt, carries a weapon, involved in gang fight, or uses a weapon in a fight; mean = 2.09; SD = 1.03). For the dimension of covert behaviors, the following three scales were created: a scale for childhood (3 items;  $\alpha = .52$ ; cheats frequently (at school), lies frequently, or frequently dishonest; mean = .67; SD = .88); a scale for early adolescence (5 items;  $\alpha = .62$ ; cheats frequently, lies frequently, burglary, major theft, or minor theft; mean = 2.24; SD = 1.54); and a scale for late adolescence (5 items,  $\alpha = .78$ ; joyriding, burglary, major theft, minor theft, or sells stolen goods; mean = 1.76; SD = 1.31).

For the dimension of reckless behavior, the following two scales were created: a scale for early adolescence (7 items;  $\alpha = .65$ , drives without a license, drinks alcohol, trespasses, watches pornographic material, goes to pubs, smokes regularly, or gambles often; mean = 2.61; SD = 1.90) and a scale for the period of late adolescence (6 items,  $\alpha = .59$ ; smokes regularly, gambles often, high sexual activity, heavy drinker, drives after drinking, or motoring offense; mean = 2.25; SD = 1.60). The Cambridge Study did not include enough information on reckless behavior during childhood to create a scale. Finally, the following two scales were used to measure authority–conflict behavior: a scale for childhood (4 items;  $\alpha = .58$ ; truancy, persistently late at school, disobedient, or difficult to discipline; mean = .52; SD = .89) as well as



a scale for early adolescence (4 items;  $\alpha = .66$ ; difficult to discipline, resentful to criticism, disobedient, or persistently late at school;  $\text{mean} = .71$ ;  $\text{SD} = 1.10$ ). The lack of behavioral indicators consistent with the dimension of authority–conflict at age 18 did not allow for the creation of such a scale for late adolescence. Higher scores on each of the ten scales indicate a higher rate of antisocial behavior.

## IPV

The measure IPV was based solely on self-reported data collected from the male's partner<sup>4</sup>. To this end, a revised version of the Conflict Tactics Scale (CTS; Straus, 1990) was used<sup>5</sup>. The CTS includes a total of the following 15 items: three related to psychological abuse, four related to minor physical violence, and eight related to major physical violence. Female partners<sup>6</sup> were asked to report about their male partner's perpetration (man-to-woman violence) as well as their male partner's victimization (woman-to-man violence) in the last 5 years<sup>7</sup>. All items were coded as dichotomous scores (0 = absent; 1 = present). A meta-analysis based on the

---

<sup>4</sup> Official data on IPV were not available at the time of the study. We would expect that only a small fraction of the men would be convicted of IPV. For example, we can extrapolate from the Dunedin Longitudinal Birth Cohort of about 500 men, who were followed to age 32, with official records of conviction that include the sort of minor nonwounding assault on a woman. Although self-report data show that the prevalence of IPV was substantial, only 6 men have an official record of "assault on a domestic female victim" (Terrie E. Moffitt, personal communication, 2008).

<sup>5</sup> This study examined a snapshot of the relationship history (past 5 years). Therefore, the current study does not provide a complete picture of IPV throughout adulthood (i.e., onset, stability, escalation, etc.). Although interesting, comparing men on their relationship history would be another study in itself, which is something that has been achieved rarely and often for brief follow-up periods. Had we looked at the relationship history throughout adulthood, a long recall period would have been necessary, which could have introduced other biases because of poor memory recall.

<sup>6</sup> Although it would have been interesting to include information for both partners, results were not available for the male partners. This lack of information may constitute a limitation to our study, but it should be understood in the context that this would have created other possible biases, which include 1) men tend to underreport IPV compared with women and 2) the same source of information (men's report) would have been used for some independent variables and the dependent variable. It should be noted that, based on previous studies (Moffitt et al., 1997), up to 80 percent of the male partner's report can be accounted for by the female's account.

<sup>7</sup> We recognize that the 5-year recall represents a strength as well as a limitation of the study. We acknowledge the fact that a 1-year recall window would have limited possible recall biases of the informant. However, a 1-year recall period would not allow us to identify longer and more serious patterns of abuse and violence. Also, serious forms of violence are rare events, and a shorter recall period would have limited the full extent of IPV behaviors. Furthermore, our study focus is not on the immediate, contextual, and situational precursors of IPV, where a 1-year period would be more appropriate, but on the propensity for men to be involved in IPV in midlife.

CTS<sup>8</sup> has shown evidence of male underreporting using self-reports compared with partner's report (Archer, 1999). Taking this into consideration, female partners completed the CTS questionnaire. The CTS was completed for 202 of the 224 (90.2 percent) female partners included in the study. In total, 78 percent of the female partners reported that their male partner had committed acts of verbal aggression, 22 percent reported acts of minor physical violence, and 7 percent reported acts of major physical violence (table 1). Victimization rates for verbal aggression (88 percent), minor physical violence (31 percent), and major physical violence (10 percent) were relatively higher than male perpetration rates, with effect sizes (Cohen's *d*) ranging from .16 (major physical violence) to .40 (verbal aggression). Scores on male perpetration of violence, as well as the scores of male victimization, were summed to create two composite scales, the male perpetration scale ( $\alpha = .87$ ) and the male victimization scale ( $\alpha = .68$ ). Not surprisingly, the scores on both scales were highly correlated (Spearman's  $\rho = .61$ ,  $p < .001$ ), which suggests the high co-occurrence of both male perpetration and male victimization within the same couple, according to the woman's report. This level of correlation between perpetration and victimization is congruent with previous reports (Magdol et al., 1998). The co-occurrence of perpetration and victimization observed in our study was high for verbal aggression (Cohen's  $d = 1.47$ ) and minor physical violence (Cohen's  $d = 1.07$ ) but smaller for major physical violence (Cohen's  $d = .46$ ). Our findings, however, do not take into account the context, motive, or consequences of the abusive and violent behavior. Considering that the scores on both the perpetration and victimization scales were moderately skewed, we used log transformations for additional analyses.

---

<sup>8</sup> This instrument has high internal consistency reliability (Cronbach's  $\alpha = .89$ ) (Koenen et al., 2003), has high interpartner agreement reliability (latent correlation = .77) (Moffitt et al., 1997), and is a strong predictor of which couples in the general population experience clinically significant violence that involves injury or intervention by official agencies (Ehrensaft, Moffitt, and Caspi, 2004).

---Insert Table 1---

### Analytical Strategy

We examined the relationship between early developmental factors and later partner violence using EQS 6.0 (Bentler, 1995). For this study, partner violence was operationalized as a latent construct composed of the covariance between the score on the scale of male perpetration and the score of male victimization. By focusing on the covariance, this study aims to predict later involvement in IPV or common couple abuse and violence. To minimize the effect of non-normally distributed variables, the Satorra-Bentler coefficient was analyzed. Mardia's coefficient was used to examine potential multivariate outliers. The Non-Normed Fit Index (NNFI), the Comparative Fit Index (CFI), and the Root Mean Square Error of Approximation (RMSEA) were also reported to assess the goodness-of-fit of the models analyzed. The NNFI was chosen over the Normed Fit Index (NFI) because, compared with the latter, it penalizes for model complexity. Similarly, we reported the RMSEA because it penalizes for models lacking parsimony, which was particularly important because the analytical strategy aimed to compare different empirical explanatory models of IPV. Therefore, scores higher than .95 on both the NNFI and the CFI and scores lower than .05 on the RMSEA all indicate an overall good fitting model.

*Autoregressive modeling.* To assess the differential impact of levels of antisociality on later IPV, we used a series of autoregressive modeling (ARM) (Jöreskog, 1979)<sup>9</sup>. Typically,

---

<sup>9</sup> Before testing the research hypotheses, we analyzed the underlying latent structure of antisocial behavior across the three developmental periods. We tested two developmentally oriented models: a developmental-only model and a mixture model, including elements from both the static and the dynamic models. The developmental-only model stipulates that antisocial behavior develops in a stagelike fashion. Therefore, rather than forcing the ten measures of antisocial behavior to cluster according to the nature of deviance, they were forced into latent constructs based on the time period at which they were measured (childhood, early adolescence, and late adolescence). This model proved to be a moderate representation of the Cambridge data [ $X^2(32) = 109.5, p < .01; NFI = .86, CFI = .90, RMSEA = .08$ ]. In other words, a model that accounts for the timing of antisociality is a better representation of the development of antisociality than a general propensity model of antisociality (Bartusch et al., 1997). We then tested a mixture model recognizing both the period of development of deviance (childhood, early adolescence, late adolescence), as well as different types of deviance (i.e., overt, covert, reckless, and authority-conflict). In this model, the correlated errors were used to account for the stability of propensities over the three developmental periods. Therefore, error terms for

ARM is used to inspect for the role of time-varying covariates where a variable is regressed on itself at an earlier time point. Two aspects of ARM were important in testing the hypothesis of a differential impact of childhood antisocial behavior. First, ARM allows the inclusion of parameters to capture time-specific influences between repeated measures. The ARM was used to test the hypothesis that childhood antisociality is more predictive of later involvement in partner violence than is adolescent antisociality. Second, to avoid model misspecifications, it was important to incorporate parameters that represent both the continuity and the discontinuity of antisociality in youth. Hence, on the one hand, the level of antisocial behavior in late adolescence was regressed on the level of antisocial behavior in early adolescence, and so on. ARM thus controls for the level of antisociality for the current period as well as for the level of antisociality in the previous period. However, the ARM does not impose a structure on the repeated measures as a function of time. The ARM is therefore somewhat limited in that no inference can be made about the underlying trajectory responsible for the observed variables.

## Results

### Antisocial Behavior in Youth and IPV

First, we examined the latent correlations among the three constructs of antisociality, IPV perpetration, and victimization (figure 1; model 1). The factor loadings and errors terms are presented in appendix 1. The model was a good fit of the data [ $\chi^2(40) = 56.73$ ,  $p < .05$ ; NNFI = .94, CFI = .97, RMSEA = .05]. As expected, the three antisociality factors were positively related

---

each of the measures of authority– conflict were allowed to correlate freely, as were the error terms for each measure of reckless behavior, and so on. This model proved to be a good representation of the Cambridge data [ $\chi^2(24) = 45.0$ ,  $p < .01$ ; NFI = .92, CFI = .96, RMSEA = .05]. In fact, it proved to be a better representation than the three-factor model without the correlated errors [ $\chi^2(8) = 79.6$ ,  $p < .01$ ]. Only four correlated errors proved to be statistically significant, as follows: 1) measures of authority–conflict at ages 10 and 14 ( $r = .40$ ,  $p < .01$ ); 2) measures of covert behavior at ages 14 and 18 ( $r = .20$ ,  $p < .05$ ); 3) measures of reckless behavior at ages 14 and 18 ( $r = .18$ ,  $p < .05$ ); and 4) measures of overt behavior at ages 10 and 14 ( $r = .15$ ,  $p < .05$ ). In sum, a model that takes into account both the timing of antisociality and, to a certain extent, the nature of antisociality proved to be the best representation of the Cambridge data. This last model was retained for additional statistical analyses.

to a higher level of partner violence perpetration and victimization. Only two of the three factors of antisociality, however, were significantly correlated with IPV, that is, antisociality in early adolescence ( $\beta = .26, p < .05$ ) and late adolescence ( $\beta = .32, p < .05$ ). Therefore, higher levels of antisocial behavior in adolescence, rather than in childhood, were associated with a higher level of IPV in adulthood. Next, structural equation modeling helped to determine which of the three antisociality factors best predicted partner violence in adulthood (Figure 1; model 2). The factor loadings and errors terms of the model are presented in appendix 1. Again, the model was a good fit of the data [ $\chi^2(41) = 57.33, p < .05$ ; NNFI = .95, CFI = .97, RMSEA = .05]. The level of antisociality in early childhood was inversely related to IPV, whereas the levels of antisociality in early and late adolescence were positively related to IPV. Only the level of antisociality in late adolescence (i.e., age 18) was a significant predictor ( $\beta = .23, p < .05$ ), which explains 12 percent of the variance in partner violence in adulthood<sup>10</sup>. It is important to note that the construct of antisociality in early adolescence was marginally related to IPV ( $\beta = .22, p < .10$ ), which raises some questions as to whether its relationship with IPV was significantly different than the one that involved late adolescence. To determine whether the construct of antisociality in late adolescence was a better predictor of IPV than the construct of antisociality in early adolescence, we constrained both paths linking the latent constructs of antisociality in early and late adolescence and IPV to be equal (model not shown). The model with the constraints added presented a good fit of the data [ $\chi^2(41) = 57.40, p < .05$ ; NNFI = .90, CFI = .97, RMSEA = .04],

---

<sup>10</sup> It could be reasonably argued that the three antisocial latent constructs are not uniform in content, which could account for the differential finding showing the superiority of latent constructs of antisociality in adolescence over and above the latent construct of antisociality in childhood. Only a study that uses the same instrument over successive developmental periods with the same research participants could answer this conundrum. To inspect for this to a certain extent, we ran the structural equation models with the same set of antisocial behaviors across the three developmental periods. In other words, each of our latent constructs of antisociality in childhood, early adolescence, and late adolescence were operationalized with only two measured variables: overt and covert antisocial behavior. The results observed were similar to those presented in the text, in that the latent constructs of antisocial behavior in adolescence were more important than of childhood.

while explaining 12 percent of the variance in IPV. Furthermore, the goodness-of-fit was equivalent to the model without the constraints [ $\chi^2(1) = 0.67, p = ns$ ], which suggests that the two constructs of antisociality in adolescence were relatively equivalent in predicting IPV in adulthood.

---Insert Figure 1---

### Childhood Risk Factors and IPV

Building on this last model, we included the measures related to neuropsychological deficits and criminogenic environment in order to determine their impact on IPV in adulthood. In a preliminary set of standard error of measurement analyses, we examined the correlations between the childhood developmental risk factors and IPV (model not shown here). Of the seven childhood developmental risk factors, the following five were significantly related to IPV in adulthood: low verbal IQ ( $r = .25, p < .01$ ), low verbal reasoning ( $r = .21, p < .01$ ), low family income ( $r = .19, p < .05$ ), parental conflict ( $r = .17, p < .05$ ), and inadequate parental skills ( $r = .14, p < .05$ ). In other words, those involved in IPV in adulthood were more likely to have shown low verbal skills and a low verbal IQ in childhood, and their family of origin was characterized by low income, parental conflict, and inadequate parenting skills. All of these results were in the expected direction.

First, we tested a baseline model to examine the indirect impact of childhood developmental risk factors on IPV through their influence on the development of antisocial behavior (figure 2; model 1). Four parameters were established, as follows: 1) developmental risk factors were allowed to correlate freely together, considering that previous studies had shown that they tended to co-occur within the same families<sup>11</sup>; 2) developmental risk factors were allowed to

---

<sup>11</sup> The following significant correlated errors were added to the models tested. In total, the following five correlated errors included family risk factors only: inadequate parenting and parental conflict ( $r = .25, p < .05$ ), low income and

correlate freely with the construct of antisocial behavior in childhood—only correlations were investigated here because both the risk factors and the antisocial behavior were measured around the same developmental time period; 3) paths between the developmental risk factors and the two latent constructs of antisociality in adolescence were added to investigate their role in the development of antisociality beyond the childhood period; and 4) considering our findings linking the antisociality constructs and IPV, a path between the late adolescence antisociality construct and partner violence construct was also added. The model, which includes the indirect impact of childhood risk factors on IPV in adulthood, yielded a good fit of the data [ $\chi^2(105) = 127.13, p < .07; NNFI = .93, CFI = .95, RMSEA = .03$ ].<sup>12</sup> Five of the seven risk factors were significantly related to antisocial behavior in childhood: low verbal reasoning ( $r = .40, p < .01$ ), low verbal IQ ( $r = .28, p < .01$ ), low SES ( $r = .28, p < .01$ ), convicted parent ( $r = .23, p < .05$ ), and inadequate parenting ( $r = .19, p < .05$ ). After we partialled out the effect of childhood antisocial behavior, which was strongly related to antisocial behavior in early adolescence ( $\beta = .57, p < .05$ ), only one developmental factor was statistically significant: low verbal IQ ( $\beta = -.18, p < .05$ ), which was inversely related to involvement in antisociality in early adolescence. Furthermore, after partialling out the effect of antisocial behavior in early adolescence, which

---

parental conflict ( $r = .24, p < .05$ ), low income and inadequate parenting ( $r = .20, p < .05$ ), low SES and low income ( $r = .18, p < .05$ ), as well as low income and convicted parent ( $r = .17, p < .05$ ). All those correlations were in the expected direction—that is, the presence of a family risk factor was significantly related to the presence of another one. One correlated error included the two individual risk factors: low verbal IQ and low verbal reasoning ( $r = .40, p < .01$ ). Also, the following five correlated errors involved family risk factors and individual risk factors: low verbal IQ and inadequate parenting ( $r = .17, p < .05$ ), low verbal IQ and low income ( $r = .34, p < .01$ ), low verbal reasoning and convicted parent ( $r = .25, p < .01$ ), low verbal reasoning and parental conflict ( $r = .18, p < .05$ ), as well as low verbal reasoning and low income ( $r = .27, p < .01$ ). Two points are of importance here: First, the strength of association of the significant correlated errors was generally low to modest, and second, verbal reasoning and verbal IQ did not share the same correlates. Those two findings suggested against the merging of the risk factors into two broad categories—that is, individual risk factors and family risk factors.

<sup>12</sup> The factor loadings and the error terms are not reported here but can be obtained from the first author. Note that the correlations between the parameter estimates obtained from the model 1 presented in figure 1 (see appendix 1 for the presentation of factors loadings and error terms) and the ones obtained from the model including the seven childhood risk factors was extremely high [ $r(32) = .99, p < .001$ ]. In other words, the parameter estimates were robust even after the inclusion of the seven risk factors into the model.

was strongly related to antisociality in late adolescence, only one risk factor emerged as statistically significant: a convicted parent ( $\beta = .20, p < .05$ ), which was positively related to the antisociality construct. The construct of antisociality in late adolescence was in turn associated positively and significantly with IPV in adulthood ( $\beta = .27, p < .05$ ). This model explained a total of 13 percent of the variance in partner violence.

---Insert Figure 2---

After establishing this baseline model, we added paths among all ten developmental risk factors and IPV in adulthood to examine the direct impact of early developmental risk factors on IPV (figure 2; model 2). Four interesting results emerged. First, the model proved to be an excellent fit of the data [ $\chi^2(98) = 112.70, p < .15$ ; NNFI = .95, CFI = .97, RMSEA = .03], and it was a statistically significant improvement over the previous model [chi square(7) = 14.43,  $p < .05$ ]. In other words, the addition of the developmental risk factors helped to predict involvement in IPV in adulthood. Second, even after adding the role of the ten developmental risk factors to the equation, the construct of antisociality in late adolescence remained statistically related to IPV ( $\beta = .25, p < .05$ ), and the direction and strength of the relationship was unchanged compared with that of the previous model. Third, after partialling out the effect of antisociality in late adolescence, only one developmental risk factor emerged as a significant predictor of IPV: low verbal IQ ( $\beta = .20, p < .05$ ). In other words, low verbal IQ measured in childhood had a direct impact on involvement in IPV more than three decades later. Men involved in IPV in adulthood had lower verbal abilities in childhood and were more likely to show elevated involvement in antisocial behavior in late adolescence. Fourth, when including the developmental risk factors in the equation, the explained variance of IPV increased from 13 percent to 20 percent.



## Discussion

The current study is one of the first to investigate the link between developmental risk factors and IPV over four decades using a prospective longitudinal framework. We address the methodological limitations of previous scientific literature by using a multimethod, multi-informant approach to improve the understanding of the link between the development of antisocial behavior in youth and IPV in adulthood. This empirical investigation contributes to the field by providing new insights into the prevalence of IPV in middle adulthood, the link between early antisociality and IPV, the unique contribution of childhood risk factors, and the ability to predict later involvement in IPV four decades after the risk factors were first measured.

### Prevalence of IPV in Midlife

Previous prospective longitudinal studies emphasized the importance of IPV in young adulthood. The current study findings suggest that the phenomenon remains prevalent beyond that period. In fact, this study reveals important findings for understanding the development of IPV in the Cambridge men. First, prevalence rates for perpetration and victimization are somewhat consistent overall with those reported in earlier investigations with young adults (Ehrensaft et al., 2003; Lavoie et al., 2002; Magdol et al., 1997). Note that our longer recall period (i.e., 5 years) in comparison with the one used in earlier prospective longitudinal studies (e.g., 1 year) might have inflated our prevalence rates compared with those of earlier studies. Most of the violence perpetrated by the Cambridge men in their mid-40s involved relatively minor acts, whereas the most serious forms of physical aggression (e.g., beating, choking, etc.) were rare. Much higher rates of perpetration of physical aggression have been reported in the Oregon study (45%; Capaldi et al., 2001). The fact that the Oregon study was based on a sample of young men at risk for delinquency reinforces our conclusion of a link between early antisociality and partner violence. Second, the prevalence of female perpetrators of aggression

and violence toward their male partner was higher than that of men toward their female partner, although the effect size was much smaller for more serious forms of aggression. The gender differences reported here were somewhat smaller than those reported elsewhere (Magdol et al., 1997; Cohen's  $d$  range = .41–.69). Our results are consistent, however, with empirical results from Archer's (2000, 2002) meta-analytical work with CTS data showing that 1) females are more likely to commit minor acts and 2) for older and married (or cohabiting) samples, gender differences in physical aggression tend to be more in the male-to-female direction. Third, considerable overlap exists between perpetration and victimization, especially for the less serious forms of abuse. Our results are congruent with the family conflict perspective, which depicts violence as mutual (Straus, 1990) and, therefore, contradictory to the stereotypical portrayal of the man as the aggressor and the woman as the victim (Felson, 2002). Our effect sizes, however, were much stronger for less serious forms of violence (i.e., verbal aggression and minor physical violence) than for more serious forms (i.e., major physical violence), which suggests that the mutuality hypothesis might apply less to cases where extreme forms of violence were committed. Fourth, and congruent with most previous studies based on community samples of men, only a minority of men were physically violent, which suggests that individual differences might play a role in the propensity to be involved in IPV in adulthood. Recall that the data used in the current study are based on women's self-reported information, which avoids possible biases attributable to men underreporting their own violence. In sum, in midadulthood, IPV seemed to be prevalent and mutual, especially for minor forms of violence, whereas more serious forms of physical aggression were uncommon and less mutual.

#### Antisocial Behavior in Youth and IPV in Adulthood

Previous longitudinal research has emphasized the importance of an early onset of antisocial behavior as an important precursor of verbal and physical aggression against an

intimate partner in young adulthood (Ehrensaft et al., 2003; Magdol et al., 1998; Woodward, Fergusson, and Horwood, 2002). In the current study, antisociality during adolescence rather than during childhood proved to be a more significant indicator of the propensity for IPV in midlife. Not only did adolescent antisocial behavior put them at risk of perpetrating violence, but also it increased their risk of being the victim of such acts from their intimate partner. The correlation observed between antisociality in childhood and IPV more than three decades later was in the predicted direction but was low and nonsignificant. Thus, contrary to Woodward, Fergusson, and Horwood's findings, our study did not find strong evidence for the direct role of an early onset of antisocial behavior on IPV. This inconsistency might be explained by the fact that Woodward, Fergusson, and Horwood's study focused on a more proximate outcome (i.e., partner violence in early adulthood) than ours (i.e., partner violence in the 40s). Another explanation may be that individuals characterized by an early onset constitute only a small subgroup of individuals, and consequently, the type of statistical analyses selected based on linear, correlational relationships did not allow finding meaningful statistical effects. To our knowledge, only one other study has compared the predictive power of involvement in antisocial behavior at different time points in youth; it found that measures in late adolescence (ages 17–18) were better predictors of physical violence in early adulthood (ages 20–23) than those in early adolescence and childhood (Capaldi et al., 2001). Even though Woodward, Fergusson, and Horwood's study demonstrated that childhood-onset offenders were more at risk of IPV than adolescent-onset offenders, the latter group was at increased risk of being involved in partner violence compared with those without a history of antisocial behavior. In other words, and congruent with our findings, those who continue to behave in an antisocial manner in adolescence, as well as those who initiate in adolescence, are at risk for later partner violence. Involvement in antisocial activities in

adolescence and associated psychosocial risks may carry over in adulthood rather than stay limited to that specific period.

#### Early Developmental Risk Factors and IPV

Previous investigations based on retrospective data stressed the role of early exposure to family conflict as an important precursor of verbal and physical aggression against an intimate partner in adulthood (Schumacher et al., 2001; Stith et al., 2004). Congruent with prospective longitudinal studies (Farrington, 1994; Lavoie et al., 2002; Magdol et al., 1998, Simons, Lin, and Gordon, 1998), we did not find strong support for a simple imitation hypothesis. More specifically, our findings supported the hypothesis that characteristics of a criminogenic family environment indirectly increase the propensity for verbal and physical aggression with an intimate partner through the development of antisociality in youth. Cambridge Study men involved in IPV were more likely to come from family environments characterized by poor parenting skills, parental conflict, and low family income. Our findings replicate those found in earlier longitudinal investigations showing the importance on the perpetration of violence of such factors as low income of the family of origin (Magdol et al., 1998), inadequate child-rearing skills, and harsh parenting practices (Lavoie et al., 2002; Simons, Lin, and Gordon, 1998; see, however, Magdol et al., 1998), and parental conflict (Ehrensaft et al., 2003). Our study suggests that for men, such factors increase the risk of being both perpetrator and victim of such acts. Socioeconomic deprivation might put pressure on the family environment, creating tension between the parents, which in turn might disrupt the quality of parenting thus favoring the emergence of antisocial behavior in the child. Consistent with earlier investigations, we also obtained findings in support of the mediational hypothesis suggesting that these three developmental risk factors may have an indirect effect on later involvement in IPV through the development of an antisocial tendency (Capaldi and Clark, 1998; Lavoie et al., 2002; Magdol et

al., 1998; Simons, Lin, and Gordon, 1998). Therefore, for the most part, children need to develop an antisocial trajectory in order for early environmental risk factors to affect negatively the youth's own intimate relationships in adulthood. Inadequate parenting skills may promote inadequate goal-oriented behaviors, whereas exposure to conflict and marital problems may foster feelings of hostility in interpersonal relationships, especially for children in families under severe socioeconomic pressure. Early socioeconomic adversities, deviant modeling, and inadequate socialization experiences may catalyze, through the development of an antisocial trajectory, the process of intergenerational transmission of IPV.

Previous investigations have somewhat neglected the possible role of early neuropsychological deficits as important precursors of verbal and physical aggression with an intimate partner. Contrary to our initial hypothesis, the presence of neuropsychological deficits, as measured by low verbal intelligence, is linked directly to the involvement in verbal and physical aggression with an intimate partner in midlife. Consistent with the scientific literature, we found significant overlap between early neuropsychological impairments, in the form of verbal deficits and behavioral problems in early childhood (Moffitt and Caspi, 2001), although the association seems to be relatively modest in nonclinical samples (Plomin et al., 2002). The fact that the effect found for low verbal IQ did not mediate the link between antisociality and IPV leaves aside the hypothesis that antisocial children might be too disruptive to benefit from intellectual stimulation, which increases their risk of later involvement in abusive and aggressive behaviors (Dionne, 2005). Children with verbal deficits may have more difficulties in successfully completing early developmental tasks associated with self-regulation and may be more limited in understanding, regulating, and communicating emotional states. It is hard to determine from a single assessment whether low verbal skills reflected stable neuropsychological deficits or a transient condition caused by a criminogenic and adverse social environment.

Consistent with earlier prospective longitudinal investigations (Magdol et al., 1998; Woodward, Fergusson, and Horwood, 2002), Cambridge Study men with low verbal IQ were more likely, four decades after being assessed, to be involved in IPV. Contrary to Moffitt's (1993) original theoretical formulation, however, we did not find a mediational effect for neuropsychological impairments through the development of antisociality; rather, our study found a direct effect. This might reflect the fact that it is not necessary for a young male with early verbal deficits to pursue an antisocial trajectory for him to be at risk of later involvement in partner violence. It might also reflect the presence of two subgroups of antisocial youths at risk of later involvement in IPV, one group being characterized by verbal skill deficits (e.g., LCP trajectory) and another not presenting such neuropsychological deficits (e.g., adolescent-limited offenders), which would be congruent with our results regarding the importance of involvement in antisocial behavior in adolescence on later involvement in IPV.

A potential mediator among antisociality, verbal deficits, and later involvement in IPV is social information processing biases. Exposure to an antisocial and violent family environment and peer group can favor the development of cognitions that encourage coercive and aggressive behaviors (Losel and Bender, 2006). From a social information standpoint, aggressive children are more likely to interpret the intentions of others as being intentionally harmful and hostile, and to believe that coercive actions may function as a viable means for obtaining or achieving a desired goal (Crick and Dodge, 1996). This process is consistent with observations that abusive men are more likely to have cognitions that support interpersonal violence and male dominance and to engage in hostile talk about women with male peers (Capaldi et al., 2001). Such biases in social information processes might disrupt intimate relationships and lead to conflicting interactions with the partner. In fact, antisocial individuals have been shown to experience less satisfaction in the forms of a lack of cohesion and consensus with their partner (Andrews et al.,

2000). The link between verbal deficits and biases in social-information processes has not been firmly established. Yet, verbal skills are an integral part of encoding, interpreting, and assessing particular situational cues relevant to social-information processing (Bennett, Farrington, and Huesmann, 2005). Such verbal deficits might increase difficulties in coping with daily life stresses for individuals who are vulnerable to negative moods and negative emotional states, especially those entrenched in an antisocial lifestyle. As shown by Moffitt et al. (2000), men characterized by high negative emotionality (i.e., lower threshold for experiencing negative emotional states and having difficulties coping with them) are more likely to perpetrate abusive behavior and to be the victim of such acts. Men's antisocial behavior, such as sexual promiscuity, gambling, alcohol abuse, and drug abuse, might exacerbate aggressive and violent reactions from the intimate partner with a history of antisociality (Magdol et al., 1998), especially if the female partner is characterized by negative emotionality and poor self-control (Moffitt et al., 2000). In sum, verbal deficits, through possible biases in social information processing deficits, might lead some individuals to use coercive tactics to intimidate and control.

#### Prediction of IPV: How Early Can We Tell?

One of the main assumptions of developmental prevention programs is the ability to screen at-risk children for intervention purposes. Results from this study suggest that secondary IPV prevention programs may be warranted for antisocial adolescents who present evidence of verbal skill deficits and who were exposed to a criminogenic family environment during childhood. Using data from the Cambridge Study, our research showed that the ability to predict verbal and physical aggression four decades after the study had started was significant, but accuracy was modest. When the early developmental risk factors measured in childhood are combined, the explained variance in IPV was 20 percent. The explanatory power of the developmental model tested in the current study was somewhat lower than what had been

observed in previous prospective longitudinal studies (i.e., 40 to 50 percent; Capaldi and Clark, 1998; Magdol et al., 1998; for an exception, see Lavoie et al., 2002). On one side, it could be argued that the modest explained variance observed in the current study is attributable to the fact that the models examined did not account for possible qualitative differences between types of men involved in IPV in adulthood<sup>13</sup>. On the other side, this hypothesis has not been controlled for in previous longitudinal studies that showed higher predictive accuracy. It is possible that the lower explained variance in our study occurs because it is based on measures derived in the 1960s when the Cambridge study first started and because of the long interval between adolescence and age 48. The higher explained variance observed in previous studies might also be attributable to IPV being measured in early adulthood (early 20s) when risk factors were more proximal and contemporary to the phenomenon being predicted. This could be explained because we did not control for developmental risk factors that might have had an impact on development in adolescence, which proved to be an important period for the risk of later involvement in IPV. In that regard, Magdol et al. (1998) found that although nearly 75 percent of the risk factors measured in adolescence were statistically related to partner violence at 21, this was true for only about 25 percent of risk factors measured in childhood. However, recall that between the start of the Cambridge study when childhood predictors were first measured (age 8) and when the

---

<sup>13</sup> Typological studies have been concerned with the presence of qualitative differences between subgroups of perpetrators. These studies have highlighted the heterogeneity of perpetrators in terms of the level of violence used against the partner, the level of violence specialization, and the type of psychopathology (Cavanaugh and Gelles, 2005; Holtzworth-Munroe and Stuart, 1994; Johnson, 1995). Taking these three factors into account, empirical evidence suggests the presence of three main types of perpetrators: 1) family-only, 2) dysphoric borderline, and 3) generally violent-antisocial (Holtzworth-Munroe and Stuart, 1994). In that regard, Lynam (2003) has argued that the generally violent-antisocial group shares many similarities with individuals following a LCP antisocial trajectory. Although empirical evidence suggests that offenders' psychopathology does vary across these types, researchers still have not firmly established whether these within-group differences do reflect qualitative differences (e.g., no psychopathology, borderline PD, or antisocial PD). In fact, researchers have argued that within-group differences observed in those studies might simply reflect a quantitative continuum (e.g., low-medium-high psychopathology) along which perpetrators differ (Capaldi and Kim, 2007; Cavanaugh and Gelles, 2005). The nature of this quantitative continuum remains open to debate. Evidence stemming from prospective longitudinal studies (including this one) suggests that this continuum could be explained, at least in part, by an antisocial propensity.



outcome was measured (age 48), 40 years had passed. Recall also that we did not control for more proximate factors (i.e., risk factors in adulthood) such as the men's socioeconomic situation, history of assault/abuse against a partner, alcohol/drug abuse/dependence, personality disorders, attitudes supporting violence against women, and prior arrest/convictions for IPV (Holtzworth-Munroe et al., 1997; Norlander and Eckhardt, 2005; Schumacher et al., 2001; Stith et al., 2004). Also, consider that the model examined both men-to-women violence and women-to-men violence. In that regard, the women's background, personality, and behavior have all influenced the outcomes studied here (Magdol et al., 1998) and none of these factors were controlled for. Furthermore, recall that we did not take into account the situational determinants, such as men/women's alcohol/drug use, stress, negative emotional states and mood, or financial/employment difficulties (Kropp and Hart, 2000). In that context, the variance explained by the developmental model analyzed in the current study is quite significant.

This study is not without methodological limitations. First, our study is based on a sample of men originally selected in the early 1960s from an inner-city working-class neighborhood in South London. Our results are as good as the indicators used when the study first started. Second, our study is based on men living with their intimate partner, and as a result, the study does not include those who were dating but not cohabitating. Hence, our results apply only to men living with their partners. Third, it is also possible that our findings reflect methodological limitations because of some disparities in our indicators of antisocial behaviors across different time points. Fourth, our study is not fully developmental as: 1) developmental changes can occur within the three periods examined; 2) we do not control for the psychosocial risk factors in other periods of development (early and late adolescence); 3) aside from antisociality, we do not address within- and between-individual changes in the psychosocial risk factors over time; and 4) we did not control for the characteristics of the participants' partners as the information was not available for

this study. Also, it would have been interesting to control for more contextual and situational factors that might have influenced involvement in IPV in adulthood.

### Conclusion

Despite these limitations, our results replicate several findings obtained with prospective longitudinal studies, which suggests many similarities and continuities between IPV in early adulthood and midlife. We find empirical evidence showing that IPV in midlife is prevalent and, therefore, is not limited to the period of early adulthood. Consistent with empirical studies based on community samples, IPV is mutual, especially for psychological abuse and minor forms of physical violence. The impact of those manifestations should not be minimized as our findings indicate that exposure to interparental conflict is correlated with later involvement in IPV. Our findings have implications for the theory development of partner violence. Of importance, the Cambridge study offered the opportunity to examine and compare the relative role of individual and familial risk factors of IPV. This study suggests that IPV is not just situational and contextual as it provides evidence that childhood risk factors measured four decades earlier were related to these manifestations. In fact, we find significant continuity between early childhood risk factors and adulthood that warrants additional scrutiny. The level of continuity observed between the development of antisocial youth and partner violence involvement in adulthood has significant policy implications. More specifically, the study findings suggest that the prevention of partner violence should start as early as adolescence. Claims have been made that such preventive efforts should start earlier by targeting children with a diagnosis of conduct disorder (Ehrensaft et al., 2003). In the current study, however, even though childhood antisociality is an important precursor of adolescent antisociality, it is not significantly linked to later partner violence. Antisociality that starts or persists in adolescence is the main risk factor identified and may be one of the key risk factors that should be targeted for the prevention of partner violence

involvement. Prevention efforts that target solely the youth's antisocial behavior may not be sufficient as we find that verbal skill deficits are also a significant risk factor of later IPV, thus suggesting the importance of a cognitive component to such interventions. Secondary prevention programs may be warranted for antisocial youths characterized by low verbal skill deficits and who were exposed to a criminogenic family environment during childhood. Furthermore, the study findings are inconsistent with claims that exposure to parental conflicts has strong negative consequences for later partner violence involvement. The results of the autoregressive modeling provide empirical evidence that indicates a need for a broader framework to tackle the problem of IPV by looking at the presence of multiple familial risk factors that characterize a criminogenic environment. This study suggests that, first, intergenerational transmission of antisocial behavior seems to explain, at least in part, the involvement in IPV. Second, results suggest that effective early prevention and intervention programs focusing on antisocial adolescents with verbal skill deficits may have an unanticipated effect on the risk of being abusive and violent later in adulthood. Third, findings are in line with a multigate preventive approach to the problem of partner violence targeting early developmental risk factors at different time points in youth. Although a conundrum remains regarding the efficacy of treatment programs with adult partner abusers, more attention should be given to lifelong benefits of early programs aimed at preventing adult antisocial behavior.

#### Funding

This research was funded by the Social Sciences and Humanities Research Council of Canada (SSHRC). The Cambridge Study was funded by the UK Home Office and the Department of Health. The authors would like to thank Raymond Corrado, Margaret Jackson, Denise Gottfredson, and three anonymous reviewers for their helpful comments on an earlier version of this article.

## References

- Andrews, Judy A., Sharon L. Foster, Deborah M. Capaldi, and Hyman Hops. 2000. Adolescent and family predictors of physical aggression, communication, and satisfaction in young adult couples: A prospective analysis. *Journal of Consulting and Clinical Psychology* 68: 195–208.
- Archer, John. 1999. Assessment of the reliability of the Conflict Tactics Scales: A meta-analytic review. *Journal of Interpersonal Violence* 14:1263–89.
- Archer, John. 2000. Sex differences in aggression between heterosexual partners: A meta-analytic review. *Psychological Bulletin* 126:651–80.
- Archer, John. 2002. Sex differences in physically aggressive acts between heterosexual partners: A meta-analytic review. *Aggression and Violent Behavior* 7:313–51.
- Babcock, Julia C., Charles E. Green, and Chet Robie. 2004. Does batterers' treatment work? A meta-analytic review of domestic violence treatment. *Clinical Psychology Review* 23:1023–53.
- Bartusch, Dawn R. J., Donald R. Lynam, Terrie E. Moffitt, and Phil A. Silva. 1997. Is age important? Testing a general versus a developmental theory of antisocial behavior. *Criminology* 35:13–48.
- Bennett, Sarah, David P. Farrington, and Ron Huesmann. 2005. Explaining gender differences in crime and violence: The importance of social cognitive skills. *Aggression and Violent Behavior* 10:263–88.
- Bentler, Peter M. 1995. *EQS Structural Equations Program Manual*. Encino, CA: Multivariate Software.
- Capaldi, Deborah M., and Sara Clark. 1998. Prospective family predictors of aggression toward female partners for at-risk young men. *Developmental Psychology* 34:1175–88.

- Capaldi, Deborah M., Thomas J. Dishion, Mike Stoolmiller, and Karen Yoerger. 2001. Physical aggression toward female partners by at-risk young men: The contribution of male adolescent friendships. *Developmental Psychology* 37:61–73.
- Capaldi, Deborah M., and Hyoung K. Kim. 2007. Typological approaches to violence in couples: A critique and alternative conceptual approach. *Clinical Psychology Review* 27:253–65.
- Caspi, Avshalom. 2000. The child is father of the man: Personality continuities from childhood to adulthood. *Journal of Personality and Social Psychology* 78:158–72.
- Cavanaugh, Mary M., and Richard J. Gelles. 2005. The utility of male domestic violence offender typologies. *Journal of Interpersonal Violence* 20:155–66.
- Cohen, Ronald A., Virdette Brumm, Tricia M. Zawacki, Robert Paul, Lawrence Sweet, and Alan Rosenbaum. 2003. Impulsivity and verbal deficits associated with domestic violence. *Journal of the International Neuropsychological Society* 9:760–70.
- Crick, Nicky R., and Kenneth A. Dodge. 1996. Social information-processing mechanisms in reactive and proactive aggression. *Child Development* 67:993–1002.
- Dionne, Ginette. 2005. Language development and aggressive behavior. In *Developmental Origins of Aggression*, eds. Richard Tremblay, Willard W. Hartrup, and John Archer. New York: Guildford Press.
- Dobash, Russell P., R. Emerson Dobash, Margo Wilson, and Martin Daly. 1992. The myth of sexual symmetry in marital violence. *Social Problems* 39:71–91.
- Ehrensaft, Miriam K., Patricia Cohen, Jocelyn Brown, Elizabeth Smailes, Henian Chen, and Jeffrey G. Johnson. 2003. Intergenerational transmission of partner violence: A 20-year prospective study. *Journal of Consulting and Clinical Psychology* 71:741–53.

- Ehrensaft, Miriam K, Terrie E. Moffitt, and Avshalom Caspi. 2004. Clinically abusive relationships in an unselected birth cohort: Men's and women's participation and developmental antecedents. *Journal of Abnormal Psychology* 113:258–70.
- Essau, Cecilia A, Satoko Sasagawa, and Paul J. Frick. 2006. Psychometric properties of the Alabama Parenting Questionnaire. *Journal of Child and Family Studies* 15:597–616.
- Fagan, Jeff, and Angela Browne. 1994. Violence between spouses and intimates: Physical aggression between women and men in intimate relationships. In *Understanding and Preventing Violence*, Volume 3: Social Influences, eds. Albert J. Reiss and Jeffrey A. Roth. Washington, DC: National Academy Press.
- Farrington, David P. 1994. Childhood, adolescent, and adult features of violent males. In *Aggressive Behavior: Current Perspectives*, ed. L. Rowell Huesmann. New York: Plenum Press.
- Farrington, David P. 2003. Key results from the first forty years of the Cambridge study in delinquent development. In *Taking Stock of Delinquency: An Overview of Findings from Contemporary Longitudinal Studies*, eds. Terence P. Thornberry and Marvin D. Krohn. New York: Kluwer.
- Farrington, David P. 2005. *Integrated Developmental and Life Course Theories of Offending*. New Brunswick, NJ: Transaction.
- Farrington, David P., Jeremy Coid, Louise Harnett, Derek Jolliffe, Nadine Soteriou, Richard Turner, and David West. 2006. Criminal Careers Up to Age 50 and Life Success Up to Age 48: New Findings from the Cambridge Study in Delinquent Development. *Research Study* 299. London, UK: Home Office.

- Feder, Lynette, and David B. Wilson. 2005. A meta-analytic review of court-mandated batterer intervention programs: Can courts affect abusers' behavior? *Journal of Experimental Criminology* 1:239–62.
- Felson, Richard B. 2002. *Violence and Gender Re-Examined*. Washington, DC: American Psychological Association.
- Fergusson, David M., Joseph M. Boden, and L. John Horwood. 2006. Examining the intergenerational transmission of violence in a New Zealand birth cohort. *Child Abuse and Neglect* 30:89–108.
- Gondolf, Edward W. 2004. Evaluating batterer counseling programs: A difficult task showing some effects and implications. *Aggression and Violent Behavior* 9:605–31.
- Gottfredson, Michael R., and Travis Hirschi. 1990. *A General Theory of Crime*. Stanford, CA: Stanford University Press.
- Holtzworth-Munroe, Amy, Leonard Bates, Natalie Smutzler, and Elizabeth Sandin. 1997. A brief review of the research on husband violence. *Aggression and Violent Behavior* 2:65–99.
- Holtzworth-Munroe, Amy, and Gregory L. Stewart. 1994. Typologies of male batterers: Three subtypes and the differences among them. *Psychological Bulletin* 116:476–97.
- Huesmann, L. Rowell, and Leonard D. Eron. 1992. Childhood aggression and adult criminality. In *Facts, Frameworks and Forecasts: Advances in Criminological Theory*, ed. Joan McCord. New Brunswick, NJ: Transaction.
- Koenen, Karestan C., Terrie E. Moffitt, Avshalom Caspi, Alan Taylor, and Shaun Purcell. 2003. Domestic violence is associated with environmental suppression of IQ in young children. *Development and Psychopathology* 15:297–311.
- Kropp, Randy, and Stephen Hart. 2000. The spousal assault risk assessment (SARA) guide: Reliability and validity in adult male offenders. *Law and Human Behavior* 24:101–18.

- Johnson, Michael P. 1995. Patriarchal terrorism and common couple violence: Two forms of violence against women. *Journal of Marriage and the Family* 57:283–94.
- Jöreskog, Karl G. 1979. Statistical estimation of structural models in longitudinal developmental investigations. In *Longitudinal Research in the Study of Behavior and Development*, eds. John R. Nesselroade and Paul B. Baltes. New York: Academy Press.
- Lahey, Benjamin B., and Irwin D. Waldman. 2003. A developmental propensity model of the origins of conduct problems during childhood and adolescence. In *Causes of Conduct Disorder and Juvenile Delinquency*, eds. Benjamin B. Lahey, Terrie E. Moffitt, and Avshalom Caspi. New York: Guilford Press.
- Lavoie, Francine, Martine Hebert, Richard Tremblay, Frank Vitaro, Lucie Vezina, and Pierre McDuff. 2002. History of family dysfunction and perpetration of dating violence by adolescent boys: A longitudinal study. *Journal of Adolescent Health* 30:375–83.
- LeBlanc, Marc. 2005. An integrative personal control theory of deviant behavior: Answers to contemporary empirical and theoretical developmental criminology issues. In *Integrated Life Course and Developmental Theories of Offending*, ed. David P. Farrington. New Brunswick, NJ: Transaction.
- LeBlanc, Marc, and Christiane Bouthillier. 2003. A developmental test of the general deviance syndrome with adjudicated girls and boys using hierarchical confirmatory factor analysis. *Criminal Behaviour and Mental Health* 13:81–105.
- Loeber, Rolf, and Marc LeBlanc. 1990. Toward a developmental criminology. *Crime and Justice* 12:375–473.
- Losel, Friedrich, and Doris Bender. 2006. Risk factors for serious and violent antisocial behaviour in children and youth. In *Dangerous Behavior, Difficult Decisions: Meeting the*



*Needs of Children Who Harm*, eds. Ann Hagell and Renuka Jeyarajah-Dent. London, UK: Jessica Kingsley.

- Lynam, Donald R. 2003. Looking earlier in the life course for the GVA: Comment on Holtzworth-Munroe and Meehan. *Journal of Interpersonal Violence* 19:1401–4.
- Magdol, Lynn, Terrie E. Moffitt, Avshalom Caspi, Jeffrey Fagan, and Phil A. Silva. 1997. Gender differences in partner violence in a birth cohort of 21-year-olds: Bridging the gap between clinical and epidemiological approaches. *Journal of Consulting and Clinical Psychology* 65:68–78.
- Magdol, Lynn, Terrie E. Moffitt, Avshalom Caspi, and Phil A. Silva. 1998. Developmental antecedents of partner abuse: A prospective-longitudinal study. *Journal of Abnormal Psychology* 107:375–89.
- Moffitt, Terrie E. 1993. Adolescence-limited and life course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review* 100:674–701.
- Moffitt, Terrie E., and Avshalom Caspi. 2001. Childhood predictors differentiate life course persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology* 13:355–75.
- Moffitt, Terrie E., and Avshalom Caspi. 2003. Preventing the intergenerational continuity of antisocial behaviour: Implications of partner violence. In *Early Prevention of Adult Antisocial Behaviour*, eds. David P. Farrington and Jeremy W. Coid. Cambridge, UK: Cambridge University Press.
- Moffitt, Terrie E., Avshalom Caspi, Honalee Harrington, and Barry J. Milne. 2002. Males on the life-course persistent and adolescence-limited antisocial pathways: Follow-up at age 26 years. *Development and Psychopathology* 14:179–207.

- Moffitt, Terrie E., Avshalom Caspi, Robert F. Krueger, Lynn Magdol, Gayla Margolin, and Phil A. Silva. 1997. Do partners agree about abuse in their relationship? A psychometric evaluation of interpartner agreement. *Psychological Assessment* 9:47–56.
- Moffitt, Terrie E., Robert F. Krueger, Avshalom Caspi, and Jeff Fagan. 2000. Partner abuse and general crime: How are they the same? How are they different? *Criminology* 38:199–232.
- Nagin, Daniel S., and Raymond Paternoster. 2000. Population heterogeneity and state dependence: State of the evidence and directions for future research. *Journal of Quantitative Criminology* 16:117–44.
- Norlander, Bradley, and Christopher Eckhardt. 2005. Anger, hostility, and male perpetrators of intimate partner violence: A meta-analytic review. *Clinical Psychology Review* 25:119–52.
- Patterson, Gerald R. 1993. Orderly change in a stable world: The antisocial trait as a chimera. *Journal of Consulting and Clinical Psychology* 61:911–9.
- Plomin, Robert, Thomas S. Price, Thalia C. Eley, Philip S. Dale, and Jim Stevenson. 2002. Associations between behaviour problems and verbal abilities and disabilities in early childhood. *Journal of Child Psychology and Psychiatry* 43:619–33.
- Raven, John C. 1943. *The Mill Hill Vocabulary Scale*. London, UK: H.K. Lewis.
- Sampson, Robert J., and John H. Laub. 2005. A life-course view of the development of crime. *Annals of the American Academy of Political Sciences* 602:12–45.
- Schumacher, Julie A., Shari Feldbau-Kohn, Amy M. Smith Slep, and Richard E. Heyman. 2001. Risk factors for male-to-female partner physical abuse. *Aggression and Violent Behavior* 6:281–352.

- Simons, Ronald L., Kuei-Hsui Lin, and Leslie C. Gordon. 1998. Socialization in the family of origin and male dating violence: A prospective study. *Journal of Marriage and the Family* 60:467–78.
- Smithey, Martha, and Murray A. Straus. 2004. Primary prevention of intimate partner violence. In *Crime Prevention: New Approaches*, eds. Helmut Kury and Joachim Obergfell-Fuchs. Mainz, Germany: Weisser Ring.
- Stith, Sandra M., Douglas B. Smith, Carrie E. Penn, David B. Ward, and Dari Tritt. 2004. Intimate partner physical abuse perpetration and victimization risk factors: A meta-analytic review. *Aggression and Violent Behavior* 10:65–98.
- Straus, Murray A. 1990. Measuring intrafamily conflict and violence: The Conflict Tactics Scales. In *Physical Violence in American Families: Risk Factors and Adaptations to Violence in 8145 Families*, eds. Murray A. Straus and Richard J. Gelles. New-Brunswick, NJ: Transaction.
- Thornberry, Terence P. 2005. Explaining multiple patterns of offending across the life course and across generations. *ANNALS of the American Academy of Political and Social Science* 602:156–95.
- West, David J., and David P. Farrington. 1973. *Who Becomes Delinquent?* London, UK: Heineman Educational Books.
- Whitaker, Daniel J., Shannon Morrison, Christine Lindquist, Stephanie Hawkins, Joyce A. O'Neill, Angela M. Nesius, Anita Mathew, and LeRoy Reese. 2006. A critical review of interventions for the primary prevention of perpetration of partner violence. *Aggression and Violent Behavior* 11:151–66.

White, Helene R., and Cathy S. Widom. 2003. Intimate partner violence among abused and neglected children in young adulthood: The mediating effects of early aggression, antisocial personality, hostility and alcohol problems. *Aggressive Behavior* 29:332–45.

Woodward, Lianne J., David M. Fergusson, and L. John Horwood. 2002. Romantic relationships of young people with childhood and adolescent onset antisocial behavior problems. *Journal of Abnormal Child Psychology* 30:231–43.

Table 1. Perpetration and Victimization Rates of Male IPV at Age 48

Items	IPV		Test	Effect Size <sup>a</sup>	Effect Size <sup>b</sup>
	Perpetration (%)	Victimization (%)	Chi Square (1)	Co-Occurrence	Gender Difference
<b>Verbal Aggression</b>	77.6	87.6	70.74**	1.47	.40
To curse or swear at you	74.3	84.2			
Order you around	43.8	53.0			
To insult or shame You	18.3	14.9			
<b>Minor Physical Violence</b>	21.9	31.3	44.83**	1.07	.29
Push, grab, or shove partner	18.3	21.3			
Slap partner	8.4	13.9			
Shake partner	3.3	2.5			
Throw an object at partner	5.8	15.3			
<b>Major Physical Violence</b>	7.4	9.9	9.97*	.46	.16
Kick, bite, or hit partner	5.0	5.9			
Twisted partner's arm	1.0	0.0			
Throw your partner bodily	2.0	0.5			
Hit/try to hit partner with object	2.5	5.0			
Beat up partner	1.5	0.5			
Choke or strangle partner	2.0	0.5			
Threaten partner with knife/gun	1.0	0.5			
Use a knife or gun	0.5	0.0			
<b>Total CTS Score</b>			<i>t</i> (201)		
Range	0–12	0–9			
Mean (SD)	1.9 (1.9)	2.2 (1.6)	2.59*	.26	

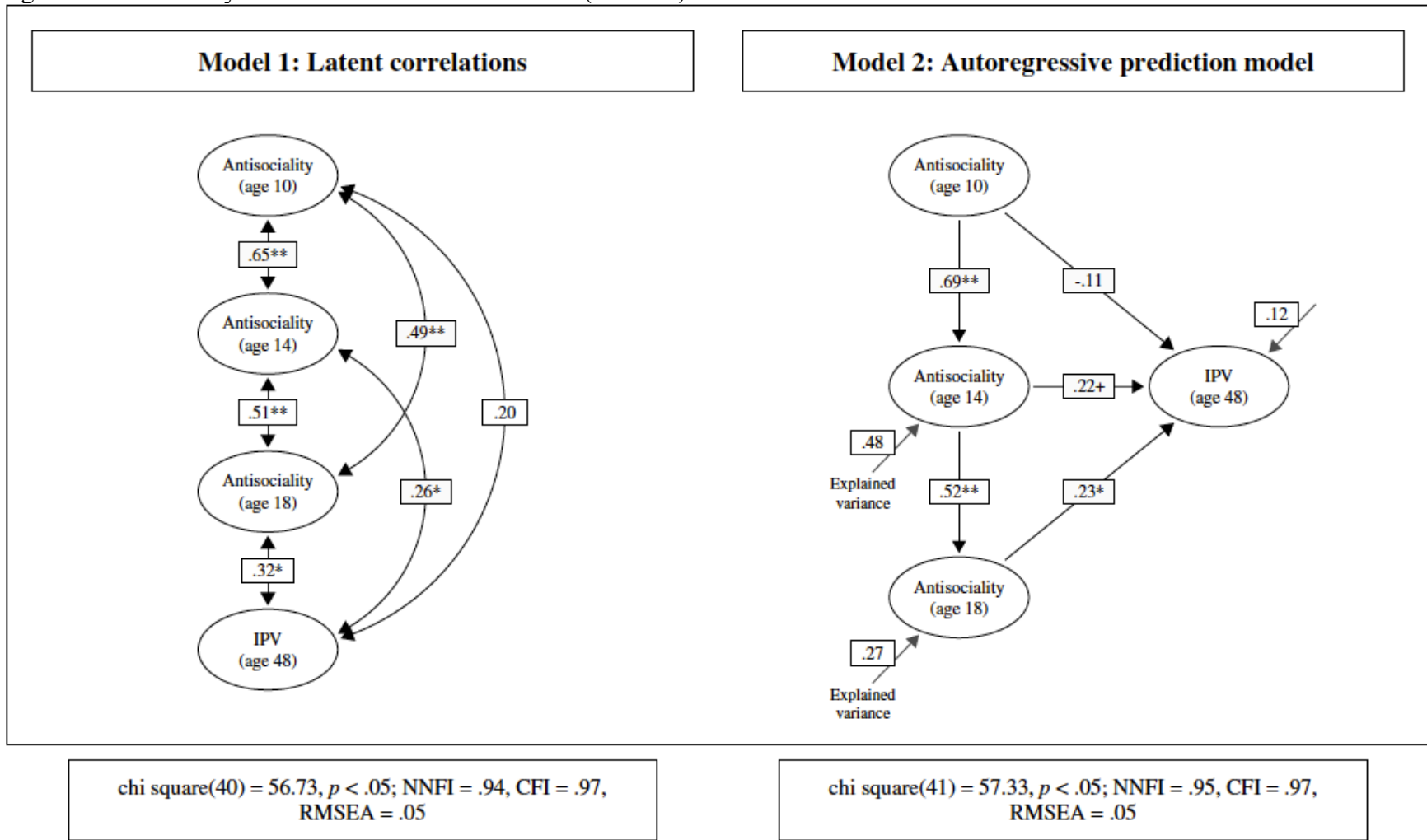
*N* = 202.

<sup>a</sup>Effect size (Cohen's *d*) measuring the co-occurrence of the same form of abuse within the intimate relationship.

<sup>b</sup>Effect size (Cohen's *d*) measuring gender differences in perpetration of abuse and violence.

\**p* < .01; \*\**p* < .001

Figure 1. Antisociality in Youth and IPV in Adulthood ( $N = 202$ )

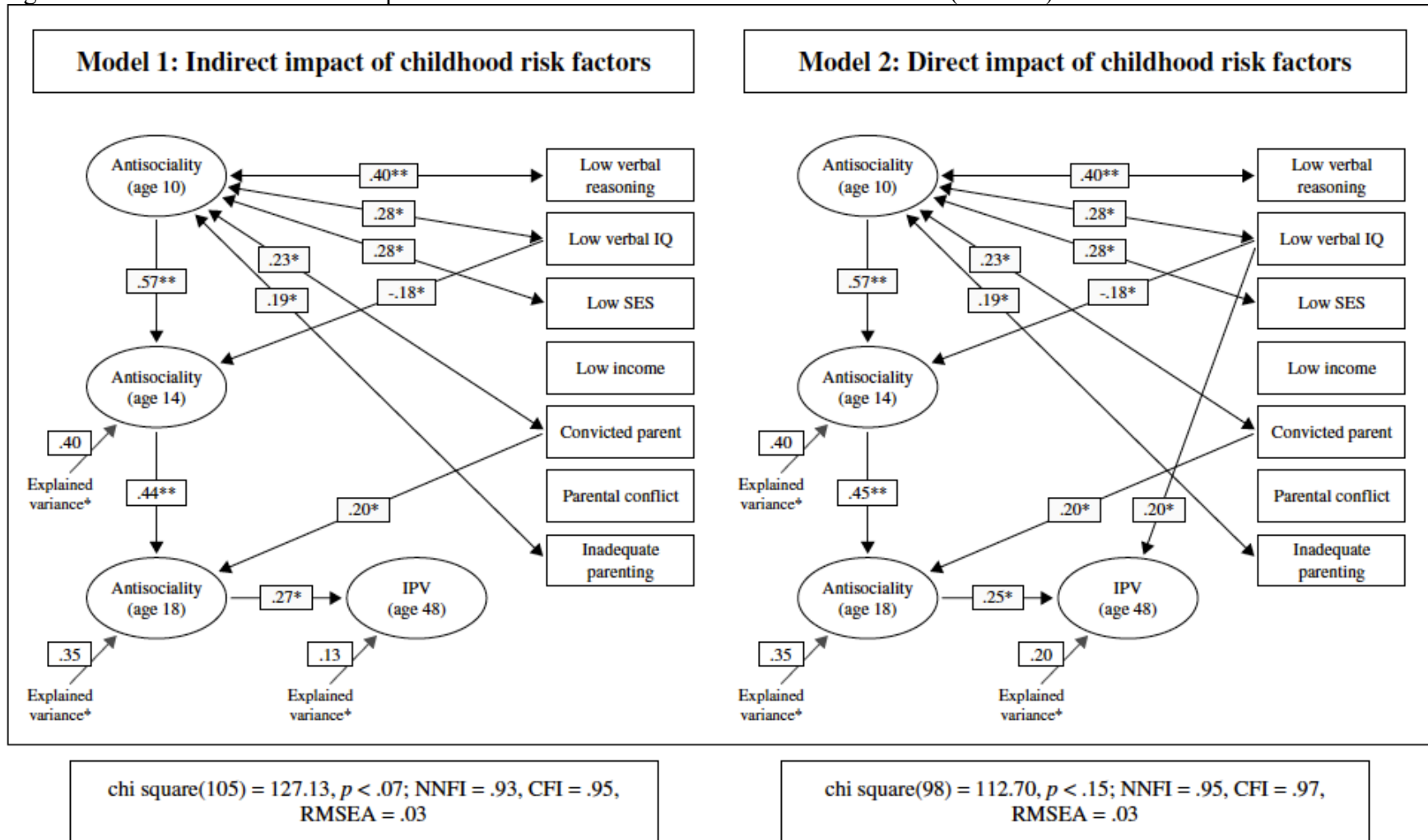


Factor loadings and correlated errors are reported in appendix 1.

Abbreviations : IPV = intimate partner violence (perpetration and victimization).

$+p < .10; *p < .05; **p < .01$ .

Figure 2. The Direct and Indirect Impact of Childhood Risk Factors on IPV in Adulthood (N = 202)



Only significant paths are reported here. Correlated errors are reported in footnote 10.

Abbreviations: IPV = intimate partner violence (perpetration and victimization).

\* $p < .05$ ; \*\* $p < .01$ .

Appendix 1

Measurement models of the link between antisocial constructs and IPV

Parameter	Model 1		Model 2	
	Parameter Estimates		Parameter Estimates	
	Loadings	Error Terms	Loadings	Error Terms
<b>Childhood Antisociality (Age 10)</b>				
Authority-conflict	.69**	.72	.64**	.77
Covert	.52**	.86	.50**	.87
Overt	.43**	.90	.45**	.89
<b>Early Adolescence Antisociality (Age 14)</b>				
Authority-conflict	.51**	.86	.54**	.85
Covert	.83**	.56	.81**	.59
Overt	.66**	.75	.68**	.74
Reckless	.66**	.75	.66**	.75
<b>Late Adolescence Antisociality (Age 18)</b>				
Covert	.56**	.83	.53**	.85
Overt	.71**	.71	.73**	.68
Reckless	.70**	.71	.67**	.74
<b>Intimate Partner Violence (Age 48)</b>				
Perpetration	.86**	.51	.85**	.53
Victimization	.72**	.69	.73**	.68
<b>Correlated Errors—Antisocial Behavior</b>				
Authority-conflict: Age 10–age 14	.50**	—	.50**	—
Covert: Age 10–age 14	.10	—	.10	—
Covert: Age 10–age 18	.02	—	.01	—
Covert: Age 14–age 18	.20*	—	.20*	—
Overt: Age 10–age 14	.10	—	.09	—
Overt: Age 10–age 18	.01	—	.01	—
Overt: Age 14–age 18	.08	—	.06	—
Reckless: Age 14–age	18.27**	—	.27**	—

Latent correlations and regression paths are reported in figure 1.

\* $p < .05$ ; \*\* $p < .01$ .