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Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females

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Abstract
This article reports a comparison on childhood risk factors of males and females exhibiting childhood-onset and adolescent-onset antisocial behavior, using data from the Dunedin longitudinal study. Childhood-onset delinquents had childhoods of inadequate parenting, neurocognitive problems, and temperament and behavior problems, whereas adolescent-onset delinquents did not have these pathological backgrounds. Sex comparisons showed a male-to-female ratio of 10 : 1 for childhood-onset delinquency but a sex ratio of only 1.5 : 1 for adolescence-onset delinquency. Showing the same pattern as males, childhood-onset females had high-risk backgrounds but adolescent-onset females did not. These findings are consistent with core predictions from the taxonomic theory of life-course persistent and adolescence-limited antisocial behavior.

Heterogeneity within a group of individuals who share a problem behavior constitutes a challenge for theory, research, and intervention design. Many students of antisocial behavior are testing whether heterogeneity within the antisocial population can be sorted out by making a distinction between problems beginning in childhood versus those beginning in adolescence. We previously described in this journal groups of childhood-onset and adolescence-onset males identified in the Dunedin birth cohort, reporting the developmental course of their antisocial behavior from age 3 years to age 18 years as well as their characteristics at age 18 years (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996). Here we describe the characteristics of the Dunedin study members and their families as measured during their childhoods. In so doing we test our hypothesis that childhood-onset, but not adolescent-onset, antisocial behavior is associated in childhood with inadequate parenting, neurocognitive difficulties, and problems of poorly controlled behavior (Moffitt, 1993). Also in this journal, Silverthorn and Frick (1999) have queried whether our taxonomy applies to girls. To respond to their query we report for the first time the characteristics of childhood-onset and adolescent-onset antisocial girls from the Dunedin cohort.

The developmental typology hypothesized that childhood-onset versus adolescent-onset conduct problems have different etiologies, and we also predicted different outcomes for the two types across the adult life course (Caspi & Moffitt, 1995; Moffitt, 1993; Moffitt, 1994; Moffitt, 1997). In a nutshell, we suggested that "life-course-persistent" antisocial behavior originates early in life, when the

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difficult behavior of a high-risk young child is exacerbated by a high-risk social environment. The child's risk emerges from inherited or acquired neuropsychological variation, initially manifested as subtle cognitive deficits, difficult temperament, or hyperactivity. The environment's risk comprises factors such as inadequate parenting, disrupted family bonds, and poverty. The environmental risk domain expands beyond the family as the child ages, to include poor relations with people such as peers and teachers. Over the first 2 decades of development a sequence of transactions between child and environment accumulate to gradually construct a disordered personality, with hallmark features of physical aggression and antisocial behavior persisting to midlife.

In contrast, we suggested that "adolescence-limited" antisocial behavior emerges alongside puberty, when otherwise healthy youngsters experience dysphoria during the relatively roleless years between their biological maturation and their access to mature privileges and responsibilities, a period we called the maturity gap. While adolescents are in this gap it is virtually normative for them to mimic the life-course-persistent youths' delinquent style as a way to demonstrate autonomy from parents, win affiliation with peers, and hasten social maturation. However, because their predelinquent development was healthy, most young people who become adolescence-limited delinquents are able to desist from crime when they age into real maturity, turning gradually to a more conventional lifestyle. This recovery may be delayed if adolescence-limited delinquents encounter factors we called snares, such as a criminal record or addiction. According to the theory, adolescence-limited antisocials are common, relatively temporary, and near normative. Life-course-persistent antisocials are few, persistent, and pathological.

This theory has since prompted discussion of its implications (e.g., Brezina, 2000; Howell & Hawkins, 1998; Lahey, Waldman, & McBurnett, 1999; Mazerolle, Brame, Paternoster, Piquero, & Dean, 2000; Osgood, 1998; Scott & Grisso, 1997; Silverthorn & Frick, 1999), and its hypotheses of two types having distinctive correlates have received empirical support from tests conducted in several samples other than the Dunedin sample (e.g., Dean, Brame, & Piquero, 1996; Kratzer & Hodgins, 1999; Nagin & Land, 1993; Nagin, Farrington, & Moffitt, 1995; Piquero, in press; Patterson, Forgatch, Yoerger, & Stoolmiller, 1998; Taylor, Iacono, & McGue, 2000; Tibbetts & Piquero, 1999; Raine, Yaralian, Reynolds, Venables, & Mednick, 2000; Roeder, Lynch, & Nagin, 1999). Some studies have reported findings partly consistent with the hypothesis of two types but have suggested useful alterations to it (e.g., Aguilar, Sroufe, Egeland, & Carlson, 2000; Fergusson, Horwood, & Nagin, 2000).

Other studies, although not necessarily presented as a test of the two types, have reported findings consonant with predictions from the taxonomy. For example, such studies have reported that measures of infant nervous-system development interact with poor parenting and social adversity to predict chronic aggression from childhood to adolescence (Arseneault, Tremblay, Boulerice, & Saucier, 2000), and violent crime (Raine, Brennan, & Mednick, 1994; Raine, Brennan, Mednick, & Mednick, 1996) but not nonviolent crime (Arseneault, Tremblay, Boulerice, Seguin, & Saucier, 2000), and that prenatal malnutrition predicts antisocial personality disorder (Neugebauer, Hoek, & Susser, 1999). Other reports show that aggressive behavior characteristic of the life-course-persistent type is highly stable (Stanger, Achenbach, & Verhulst, 1997) and heritable (Edelbrock, Rende, Plomin, & Thompson, 1995), whereas, in contrast, rule-breaking behavior characteristic of the adolescence-limited type increases between ages 10 and 17 years (Stanger et al., 1997) and is less heritable than aggression (Edelbrock et al., 1995). Reports supporting the theory of adolescence-limited delinquency show that when young people enter adolescence they begin to admire aggressive peers and find good students less attractive (Bukowski, Sippola, & Newcomb, 2000; Luthar & McMahon, 1996); adolescents' concerns about appearing immature increase their likelihood of delinquency (Zebrowitz, Andreoletti, Collins, Lee, & Blumenthal, 1998); and delinquent peers directly promote adolescence-onset delinquency, whereas parenting and behavior problems

lead to early-onset delinquents' affiliations with delinquent peers (Simons, Wu, Conger, & Lorenz, 1994; Vitaro, Tremblay, Kerr, Pagani, & Bukowski, 1997).

Differential Risk Factors for Males on the Life-Course-Persistent Versus Adolescence-Limited Paths in the Dunedin Study

Our own studies of males in the Dunedin cohort have operationalized the two prototypes of antisocial behavior using varying statistical models, including comparison groups (Moffitt, 1990; Moffitt & Harrington, 1996; Moffitt et al., 1996), repeated-measures multiple regression (Moffitt, Lynam, & Silva, 1994), and confirmatory factor analysis (Jeglum–Bartusch, Lynam, Moffitt, & Silva, 1997), and have examined both childhood predictors and adolescent outcomes. Our studies of outcomes in adolescence have shown that the life-course-persistent path is differentially associated in males with weak bonds to family, early school leaving, and psychopathic personality traits of alienation, impulsivity, and callousness (Moffitt et al., 1996), as well as conviction for violent crimes (Jeglum–Bartusch et al., 1997; Moffitt et al., 1996). In contrast, we found that the adolescence-limited path is differentially associated with a tendency to endorse unconventional values, with a personality trait called social potency (Moffitt et al., 1996), and with nonviolent delinquent offenses (Jeglum–Bartusch et al., 1997).

Our own studies of childhood predictors have shown that the life-course-persistent path is differentially predicted by undercontrolled temperament measured by observers at age 3 years (Moffitt et al., 1996), delayed motor development at age 3 years (Moffitt, 1990), low verbal ability and hyperactivity (Jeglum–Bartusch et al., 1997; Moffitt, 1990), and poor scores on neuropsychological tests (Moffitt et al., 1994). In contrast, we found that the adolescence-limited path is differentially predicted by delinquent peers (Jeglum–Bartusch et al., 1997). Although our findings for selected predictive measures are consistent with the theory's hypotheses, they constitute only a small selection from the many measures of child and family characteristics available from the early years of the Dunedin Study. Here we present for the first time a comparison of the child- and adolescent-onset study members on the study's 26 major childhood indicators that are relevant to the theory. We test the hypothesis originally specified, that the prospective predictors of life-course-persistent antisocial behavior include "health, gender, temperament, cognitive abilities, school achievement, personality traits, mental disorders (e.g., hyperactivity), family attachment bonds, child-rearing practices, parent and sibling deviance, and socioeconomic status," whereas the predictor of adolescence-limited antisocial behavior should be "knowledge of peer delinquency" (Moffitt, 1993, p. 695).

Males Versus Females

Our own tests of the theory have focused on males, prompting some to wonder whether or not females fit into the taxonomy or require a separate theory all their own (Silverthorn & Frick, 1999). The original statement of the taxonomy asserted that the theory accounts for the behavior of females as well as it accounts for the behavior of males. The full text of the theory which included predictions about females was published as a book chapter that is not widely available (Moffitt, 1994). Therefore, we quote the original statement, written in January 1991:

> The crime rate for females is lower than for males. In this developmental taxonomy, much of the gender difference in crime is attributed to sex differences in the risk factors for life-course-persistent antisocial behavior. Little girls are less likely than little boys to encounter all of the putative initial links in the causal chain for life-course-persistent antisocial development. Research has shown that girls have lower rates than boys of symptoms of nervous system dysfunction, difficult temperament, late milestones in verbal and motor development, hyperactivity, learning disabilities, reading failure, and childhood conduct problems. Thus, the consequent processes of cumulative continuity ensue for far fewer girls than boys. Most girls lack the personal diathesis elements of the evocative, reactive, and proactive person/environment interac-

tions that initiate and maintain life-course persistent antisocial behavior.

Adolescence-limited delinquency, on the other hand, is open to girls as well as to boys. According to the theory advanced here, girls, like boys, should begin delinquency soon after puberty, to the extent that they (1) have access to antisocial models, and (2) perceive the consequences of delinquency as reinforcing. . . . However, exclusion from gender-segregated male antisocial groups may cut off opportunities for girls to learn delinquent behaviors. . . . Girls are physically more vulnerable than boys to risk of personal victimization (e.g., pregnancy, or injury from dating violence) if they affiliate with life-course persistent antisocial males. Thus, lack of access to antisocial models and perceptions of serious personal risk may dampen the vigor of girls' delinquent involvement somewhat. Nonetheless, girls should engage in adolescence-limited delinquency in significant numbers. (Moffitt, 1994, pp. 39–40)

The original theory thus proposed that (a) fewer females than males would become delinquent (and conduct disordered) overall and that (b) within delinquents the percentage who are life-course persistent would be larger among males than females. Following from this, (c) the majority of delinquent females will be of the adolescence-limited type, and, further, (d) their delinquency will have the same causes as adolescence-limited males' delinquency. In contrast, Silverthorn and Frick (1999) proposed that despite the fact that girls' onset is delayed until adolescence, there is no analogous pathway in girls to the adolescence-limited pathway in boys. They argued for a female-specific theory in which all delinquent girls will have the same high-risk causal backgrounds as life-course-persistent males.

Heretofore, only three empirical tests of this taxonomy have compared how females and males fit aspects of its two developmental trajectories (Fergusson et al., 2000; Kratzer & Hodgins, 1999; Mazerolle et al., 2000). This dearth of gender comparisons originates from a pragmatic circumstance. A test of this developmental–epidemiological theory requires a representative (nonclinical, nonadjudicated) sample that is followed longitudinally from childhood with repeated measures of antisocial behavior. To date few such studies have included females in large enough numbers to study the rare phenomenon of the life-course-persistent girl with adequate power for significance testing. (The three aforementioned studies examined cohorts of 1000 to 14000 individuals.) This constraint applies to the Dunedin cohort too. We have previously described the adolescence-limited causal pathway among Dunedin sample females, showing that each girl's delinquency onset is linked to the timing of her own puberty and that delinquent peers are a necessary condition for adolescent-onset girls (Caspi, Lynam, Moffitt, & Silva, 1993; Moffitt, Caspi, Rutter, & Silva, 2001). However, we have not previously attempted to study the life-course-persistent part of the taxonomy among Dunedin females.

In this article, we explore how Dunedin females fit into the operational definition of life-course-persistent and adolescence-limited groups that we previously published for Dunedin males (Moffitt et al., 1996) and, in so doing, we pit our assertion that the taxonomy describes both males and females against the competing hypothesis that girls with adolescent-onset delinquency suffer the background risk factors of life-course-persistent males and therefore warrant a special theory of their own (Silverthorn & Frick, 1999).

Method

The birth cohort

Participants are members of the Dunedin Multidisciplinary Health and Development Study, a longitudinal investigation of health and behavior (Silva & Stanton, 1996). The cohort of 1037 children (52% male, 48% female) was constituted at age 3 years, when the investigators enrolled 91% of the consecutive births between April 1972 and March 1973 in Dunedin, New Zealand. Cohort families represent the full range of socioeconomic status in the general population of New Zealand's South Island and they are primarily White; fewer than 7% self-identify as Pacific Islanders. This report uses data from assessments at ages 3 (n = 1037), 5 (n = 991), 7 (n = 954),

9 (n = 955), 11 (n = 925), 13 (n = 850), 15 (n = 976), and 18 (n = 993) years. Rates of diagnosed conduct disorder, self-reported delinquency, and crime victimization in New Zealand are similar to those in the United States; for documentation supporting generalization from the Dunedin cohort to other settings, see Moffitt et al. (2001).

Measures of childhood risk characteristics

We present data for 26 measures taken in childhood, selected to represent the three domains of childhood risk specified by the theory: family adversity and inadequate parenting (10 measures), child neurocognitive health (8 measures), and child temperament and behavior (8 measures). In addition, we present the study's two measures of peer delinquency, taken at ages 13 and 18 years. The 28 measures are described in the Appendix. Evidence of reliability (test–retest or internal consistency) and validity in the Dunedin Study has been published for virtually all of the risk predictors, and the appropriate publications are cited in the Appendix.

Designating the comparison groups of the taxonomy

To operationalize the theory of two types, we designated comparison groups on the basis of individual life histories from age 5 years to age 18 years. The scales measuring antisocial behavior used to define the comparison groups came from the Rutter Child Scales, completed by parents and teachers when the children were ages 5, 7, 9, and 11 years, and the Self-Reported Delinquency interview administered to study members at ages 15 and 18 years. The item content, scale construction, and psychometric qualities of these 10 measures were described in detail in our earlier report about Dunedin males (Moffitt et al., 1996, this journal). All 10 measures required for classifying behavioral histories were present for 477 males and 445 females (89% of both sexes). Elsewhere we have shown that missing status for the 11% of the cohort not studied here is not systematically correlated with study measures of antisocial behavior; the study members examined here closely match the original representative cohort (Moffitt et al., 1996, 2001).

The procedure for defining the groups has been described in detail in our earlier report about Dunedin males (Moffitt et al., 1996) and therefore is only briefly summarized here. The first step of the computerized algorithm divided the sample into study members who had childhood histories of antisocial behavior problems versus those who did not. Study members were considered to be antisocial children if they had evidence of extreme childhood antisocial behavior problems that were both stable across time (at least three of the assessment occasions at ages 5, 7, 9, and 11 years) and pervasive across situations (reported by parents at home and corroborated by teachers at school). The second step divided the sample into study members who participated in many antisocial acts during midadolescence versus those who did not. Study members were considered to be antisocial adolescents if they self-reported extreme delinquency at the age-15-years interview or at the age-18-years interview. On the third step, the childhood categories were combined with the adolescent categories to yield developmental profiles. Study members who met criteria for extreme antisocial behavior across both childhood and adolescence were designated on the *life-course-persistent path,* hereafter referred to as the LCP path. Study members who met criteria for extreme antisocial behavior as adolescents, but who had not been extremely antisocial as children, were designated on the *adolescence-limited path,* hereafter called the AL path.

Results

How many females fit the AL and LCP paths?

Our earlier publication (Moffitt et al., 1996) defined groups of males using cutoff criteria that were determined on the distributions of the 10 aforementioned measures of antisocial behavior *within* males, yielding 7% of males defined as on the LCP path and 23% of males on the AL path. The present report required a

comparison of males and females defined using a single standard. Therefore, we repeated the group designation exercise using the same computerized algorithm that defined the groups in Moffitt et al. (1996), but applying uniform cutoffs calculated on the distributions of the 10 antisocial measures for the full sample, regardless of sex. These gender-neutral cutoffs yielded for the AL path 122 males (26%) and 78 females (18%). On the LCP path were 6 females (1% of females) and 47 males (10% of males, including all 32 who were on the LCP path in Moffitt et al., 1996). Thus, consistent with the expectations from the theory, the male-to-female ratio for the LCP path was 10 : 1, whereas the sex ratio for the AL path was 1.5 : 1.

Do females and males on the same subtype paths share the same risk backgrounds?

Two central predictions about gender from the theory were that (a) males and females who were members of the LCP group should share the same childhood risk factors specified by the theory and (b) males and females who were members of the AL group should share the same lack of childhood risk factors. The conventional approach to testing would be an analysis of variance with group (LCP vs. AL paths) and sex (males vs. females) as factors, comparing mean levels on a risk variable. A significant group-by-sex interaction term would attest that there were sex differences on risk factors within a subtype group. By predicting a *lack* of sex differences within types, the theory called for seeking to confirm the null hypothesis via a nonsignificant sex interaction term. However, statistical power posed a problem for significance testing. Because our theory predicted few LCP group members (we found only six females), the null hypotheses could be "confirmed" falsely as a result of low statistical power. Therefore, we did not attempt tests involving LCP females and simply present the males' and females' mean scores on risk factors for visual inspection of effect sizes in Figure 1 for the LCP path and in Figure 2 for the AL path.

Figures 1 and 2 present the mean scores of study members on the two delinquent paths on prospective study risk measures of parenting, neurocognitive difficulties, and temperament–behavior, as well as peer delinquency measured in adolescence. Table 1 gives details of group means and standard deviations, indicates group differences that could be tested, and notes which measures had mean-level differences between cohort males and females.[1] Because the groups were defined using norms for the full cohort, the figures show risk factors plotted as *Z* scores standardized on the full cohort with a mean of 0 and standard deviation (*SD*) of 1. Thus, each group's mean *Z* score indicates how far that group deviates from the mean score for the representative sample (0), a mean that can be interpreted as a normative standard. The distance in *SD* units between the group's mean and the normative zero may be interpreted as the effect

1. Across the 28 risk factors listed in Table 1, data were missing for 1% or fewer individuals on 17 risk variables, for between 1–5% of individuals on 6 variables, and for more than 5% of individuals on 5 variables: parent's conviction (9%), heart rate (9%), caregiver changes (17%), neuropsychological memory (21%), and delinquent peers at 13 years (21%). The amount of missing data did not differ among the groups ($p > .10$) for 27 of the 28 risk variables. For these 27 risk variables that had low rates of missingness and no significant association between missingness and the grouping variable, we substituted missing data with the mean score and report analyses with $N = 922$ in Table 1. We report the analysis for $N = 870$ for parents' criminal conviction because missingness was significantly associated with group; 15% of the LCP group but only 5% of the other two groups had missing data, $\chi^2 (2) = 9.86$, $N = 922$, $p < .01$, indicating that parents' crime may be underestimated for the LCP path. To double-check that missing data were not problematic, we conducted two further analyses. We repeated the Table 1 comparisons between the LCP and AL groups in a regression framework, using mean-substituted risk variables while adjusting the group contrast by a binary dummy covariate in which 0 indicated nonsubstituted data and 1 indicated mean-substituted data. We also repeated the comparisons in Table 1 using only data for study members who had present data. In both checks, we obtained the same effects as shown in this table, both in terms of significance testing and substantive interpretation. There are more complex methods to deal with missing data, but these methods have been developed for instances when the data are missing for many cases, or on many variables, or when missing cases bias the sample in a systematic way, problems that do not afflict the Dunedin Study.

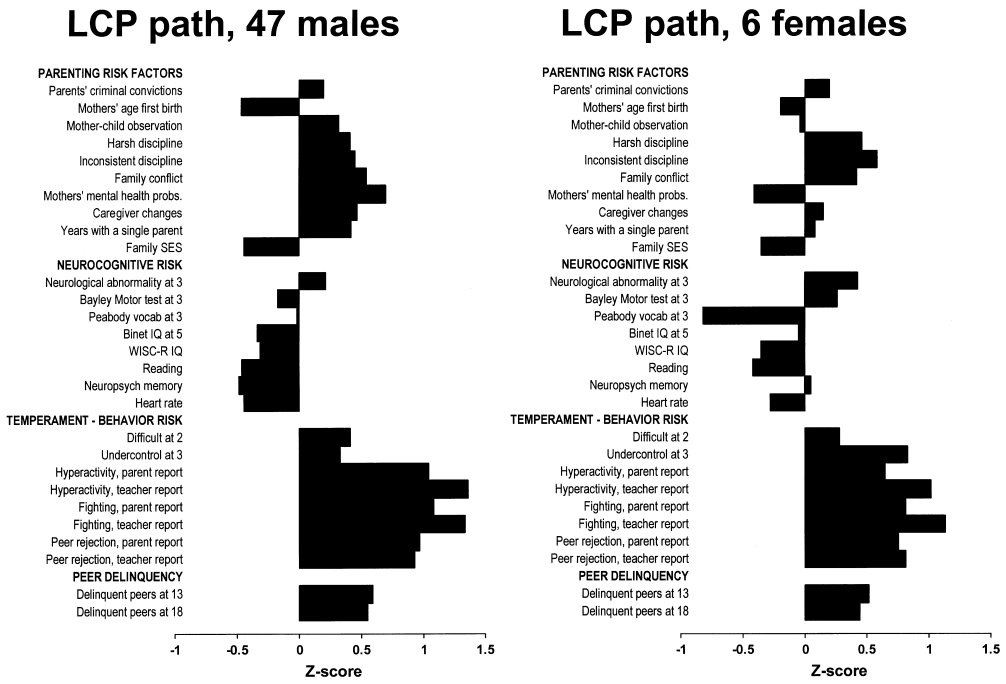

Figure 1. Mean standardized scores on risk predictors for antisocial behavior for groups of males and females on the life-course persistent path, as compared to zero, which is the norm for the birth cohort.

size, where .2 *SD* is a small effect, .5 *SD* is a medium effect, and .8 *SD* is a large effect (Cohen, 1988).

If the taxonomy applies to females as well as to males, we should observe the following three patterns in Figures 1 and 2: (a) both males and females on the LCP path should deviate from the cohort norm on the risk factors, (b) both males and females on the AL path should score near the norm ($Z = 0$) on the childhood risk factors, and (c) both males and females on the AL path should deviate from the norm on peer delinquency. Visual inspection of Figures 1 and 2 reveals that the data are generally consistent with the three patterns expected by the theory.

Consistent with the first aforementioned pattern (a), Figure 1 shows that the 47 males and 6 females on the LCP path were more similar to each other than different on most risk factors. Notably, the LCP-path girls were almost as extreme on fighting at age 5 years to 11 years relative to the full sample as were LCP-path males, confirming that these girls had problems before adolescence.[2] The pattern of means across measures within the LCP-path females was somewhat less consistent than the pattern within LCP-path males, but some of this inconsistency may be ascribed to the ease with which a variable's mean can be influenced in a group of only 6 individuals. One pattern seemed consistent enough to be noteworthy: LCP-path females stood apart from LCP-path males by having mothers whose parenting was rated by observers as average, who reported few mental health problems, and who were no more likely to be single than the average study mother.

Comparison of Figure 1 with Figure 2 shows that LCP-path members of both sexes

2. The measures of fighting on figures and tables in this article also contributed items to the omnibus childhood antisocial behavior scales that were used to define the comparison groups. However, fighting is singled out for display because of recent special interest in whether girls engage in physical fighting.

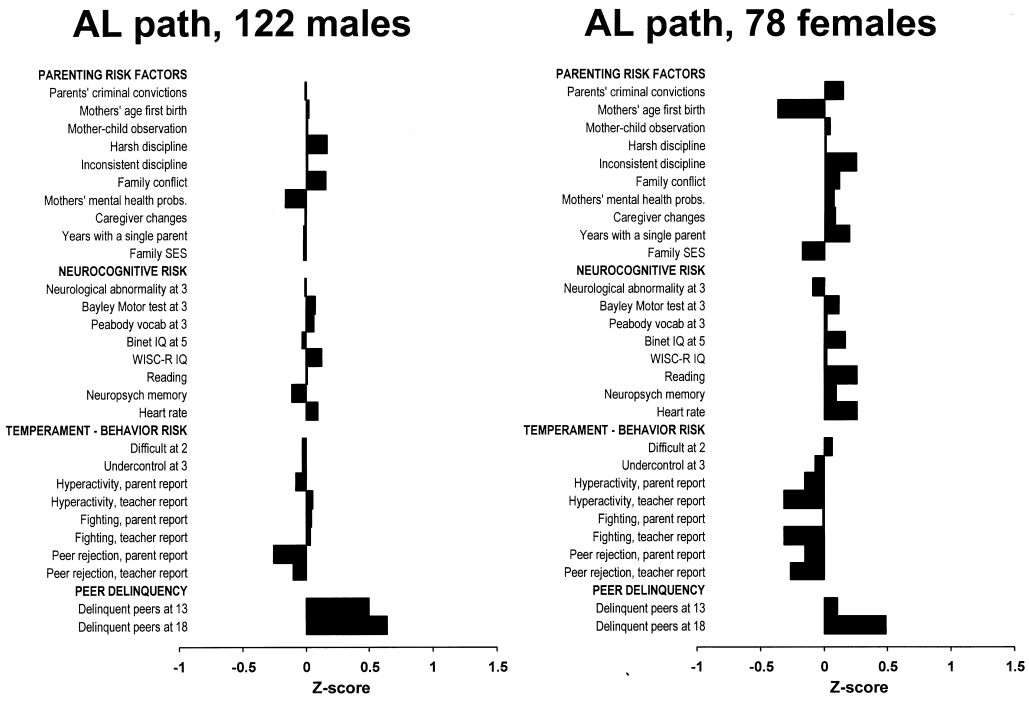

Figure 2. Mean standardized scores on risk predictors for antisocial behavior for groups of males and females on the adolescence-limited path, as compared to zero, which is the norm for the birth cohort.

tended to have worse levels of risk than their counterparts on the AL path. LCP-path males scored worse than the average study member, and worse compared to AL-path males, on every measure excepting the Peabody Picture Vocabulary test at age 3 years. Effect sizes were at least small, and many were medium. Contrasts of the difference between LCP-path males and AL-path males revealed that LCP-path males scored significantly worse on 20 of the 26 risk measures (Table 1). Likewise, LCP-path females scored worse on most of the measures than the average study member, and worse compared to AL-path females.

Consistent with the second aforementioned predicted pattern (b), both girls and boys on the AL path generally showed mean levels of risk that were near-normative for the sample. When exceptions to this normative pattern arose for AL-path males, the exceptions reflected unusually low risk (i.e., AL-path boys were *less likely* to be rejected by peers than the cohort average). When exceptions to this normative pattern arose for AL-path females, the exceptions also reflected unusually low risk. This generally occurred because there are mean level sex differences on the risk factor (i.e., relative to the sample norm AL-path girls were better readers, less hyperactive, and less likely to fight because girls in general score better than boys on these variables; Table 1). Contrasts revealed that AL-path females did not score significantly worse than AL-path males on any of the 26 risk measures excepting one: AL-path females had mothers who were younger the first time they gave birth (Table 1). However, these adolescent-onset girls' backgrounds did not otherwise resemble the high-risk backgrounds of childhood-onset males. Contrasts of the difference between AL-path females and LCP-path males revealed that AL-path females scored significantly better on 19 of the 26 risk measures (Table 1).

Consistent with the third predicted pattern (c), AL-path offenders, regardless of their sex, knew a lot more delinquent peers than the average Dunedin cohort member.

Do the backgrounds of childhood- and adolescent-onset delinquents differ significantly?

Given the absence of striking sex differences in the ways that background risk factors were associated with the LCP and AL paths, as shown in Figures 1 and 2, we collapsed the groups across sex to gain statistical power for testing differences between the LCP- and AL-path groups. This analysis parallels that presented in this journal by Aguilar et al. (2000). Results from the comparisons are shown in Table 2. Table 2 also shows for comparison purposes the group means for the 669 study members who were not classified into the AL or LCP paths, hereafter referred to as the unclassified group.[3]

Before asking whether there are group differences on risk background, it is important to establish that the LCP-path and AL-path groups showed similar levels of participation in delinquency as adolescents. This is important because the theory specifies that LCP and AL types have *different* mean levels of risk factors despite exhibiting *similar* mean levels of delinquent offending. Table 2 begins by showing each group's mean variety of different illegal acts committed at least once in the past year, separately by sex. The top two rows of Table 2 (for males) reveal that the LCP- and AL-path males were well matched on offending at ages 15 and 18 years, and that both LCP- and AL-path males offended more than unclassified males. (The two rows for males also show the expected increase in offending from age 15 years to age 18 years among males.) We have previously shown that the two path groups did not differ during adolescence on police arrests or court convictions (Moffitt et al., 1996). The next two rows of Table 2 (for females) reveal that the LCP and AL females were also well matched on offending at age 15 years. Both female groups matched both male groups as well; all four delinquent groups reported approximately seven to eight different offense types at age 15 years. Although LCP-path and AL-path females differed from each other at age 18 years, both groups offended more than the unclassified females, at both ages. (The two rows also show the oft-reported female pattern of a peak in delinquent involvement at age 15 years followed by a decrease by age 18 years; for a review of studies see Moffitt et al., 2001.) Thus, study members on the LCP and AL paths were well matched on levels of antisocial involvement as teens.

The remaining rows of Table 2 present the mean scores on the study risk measures for unclassified study members and those on the two delinquent paths. Consistent with the hypothesis that LCPs have worse backgrounds than ALs, the difference between the LCP-path group and the AL-path group on the 26 childhood risk factors (shown in the last column) was a small effect (SD = .2–.49) for 15 risk factors, medium (SD = .5–.79) for 3 risk factors, and large (SD > .8) for 6 risk factors. Group comparisons were t tests with alpha set at $p < .05$ (shown in the fifth and sixth columns). These indicated that children on the LCP path experienced significantly worse risk than children on the AL path on 21 of the 26 risk factors. In addition, small effect sizes were observed for mother–child observation, years with a single parent, and the Bayley motor score, but the differences did not reach significance at .05.

Consistent with the hypothesis that ALs have average backgrounds, the difference between the AL-path group and the cohort norm (shown in the third column) was less than a small effect (i.e., $SD < .2$) for 25 of the 26 childhood risk factors. The AL-path group scored slightly worse (mean effect size: SD = .07) than the norm on half of the variables but slightly better than the norm on the other half of the variables (mean effect size: SD = .09). Peer rejection was the sole risk factor to reach

3. The unclassified group (n = 669, 46% male) comprises (a) study members whose antisocial behavior had been too normative from age 5 to 18 years to meet criteria for the AL or LCP groups (n = 504, 48% male), (b) study members who had abstained from antisocial behavior on all measures from age 5 to 18 years (n = 92, 27% male), and (c) study members who suffered pervasive and persistent serious childhood antisocial behavior but whose adolescent delinquency was not extreme enough to meet criteria for the AL or LCP groups (n = 73, 55% male).

Table 1. *Male and female Dunedin Study members on the life-course-persistent and adolescence-limited paths, compared on risk factors for delinquency*

	z Score ($M \pm SD$)				Contrast[b]		
	Life-Course Persistent		Adolescence-Limited				
	Males ($N = 47$)	Females ($N = 6$)	Males ($N = 122$)	Females ($N = 78$)	LCP Males vs. AL Males	AL Females vs. AL Males	LCP Males vs. AL Females
Risk factors (age in years)							
Parents' criminal conviction	.20 ± 1.1	.20 ± 1.3	−.01 ± 1.0	.15 ± 1.1			
Mother's age at her first birth	−.47 ± 0.9	−.20 ± 1.2	.02 ± 1.1	−.36 ± 0.9	a	c	
Mother–child observation (3)	.32 ± 1.1	−.04 ± 1.0	.01 ± 0.9	.05 ± 1.3	a		
Harsh discipline (7–9)[a]	.41 ± 0.9	.46 ± 1.2	.17 ± 1.2	.01 ± 1.1			d
Inconsistent discipline (7–9)	.45 ± 1.1	.58 ± 1.7	.01 ± 0.9	.25 ± 1.0	a		
Moos family conflict (7–9)	.54 ± 1.1	.42 ± 0.9	.16 ± 1.0	.12 ± 1.0	a		d
Mother's mental health (7–11)	.70 ± 1.2	−.41 ± 0.7	−.16 ± 0.8	.08 ± 1.0	a		d
Caregiver changes (birth–11)	.47 ± 1.3	.15 ± 1.2	.00 ± 1.0	.09 ± 1.0	a		d
Years single parent (birth–11)	.37 ± 1.3	.12 ± 1.0	−.02 ± 0.8	.20 ± 1.2	a		
Family SES (birth–15)	−.44 ± 0.9	−.35 ± 1.0	−.02 ± 1.0	−.17 ± 1.0	a		
Child neurocognitive risk factors (age in years)							
Neurological abnormality (3)[a]	.21 ± 1.2	.43 ± 1.8	−.01 ± 0.9	−.09 ± 0.9			d
Bayley motor test (3)	−.17 ± 1.0	.26 ± 0.7	.08 ± 0.8	.12 ± 0.8			d
Peabody Vocabulary (3)	−.02 ± 0.9	−.82 ± 0.8	.07 ± 1.0	.02 ± 0.9			
Binet IQ (5)[a]	−.34 ± 0.8	−.05 ± 1.2	−.03 ± 1.0	.17 ± 0.9	a		d
WISC-R VIQ (7,9,11)	−.31 ± 0.9	−.35 ± 1.0	.13 ± 1.0	.02 ± 0.9	a		d
Reading (7,9,11)[a]	−.46 ± 1.0	−.42 ± 0.9	.01 ± 1.0	.26 ± 0.9	a	b	d
Neuropsych memory (13)[a]	−.48 ± 1.2	.05 ± 0.9	−.11 ± 1.0	.10 ± 1.0	a		d
Heart rate (7,9,11)[a]	−.46 ± 1.0	−.42 ± 0.9	.01 ± 1.0	.26 ± 0.9			d

Child temperament–behavior risk factors (age in years)							
Difficult to manage (2)	.41 ± 0.9	.28 ± 0.0	−.03 ± 1.0	.07 ± 0.9	a		d
Under control observed (3)[a]	.33 ± 1.2	.83 ± 2.2	−.03 ± 0.9	−.07 ± 1.0	a		d
Hyperactive, parent (5–11)[a]	1.04 ± 1.1	.65 ± 1.2	−.08 ± 1.0	−.15 ± 0.9	a		d
Hyperactive, teacher (5–11)[a]	1.36 ± 1.1	1.02 ± 1.5	.06 ± 0.9	−.31 ± 0.7	a	b	d
Fighting, parent (5–11)[a]	1.08 ± 0.9	.81 ± 0.7	.05 ± 0.9	−.01 ± 0.8	a		d
Fighting, teacher (5–11)[a]	1.34 ± 1.2	1.13 ± 0.9	.03 ± 0.9	−.31 ± 0.7	a	b	d
Peer rejection, parent (5–11)	.97 ± 1.3	.75 ± 1.6	−.25 ± 0.7	−.15 ± 0.7	a		d
Peer rejection, teacher (5–11)	.93 ± 1.4	.81 ± 0.8	−.10 ± 0.8	−.26 ± 0.7	a		d
Peer delinquency in adolescence (age in years)							
Delinquent peers (13)[a]	.59 ± 1.3	.52 ± 1.0	.50 ± 1.0	.11 ± 1.0		b	d
Delinquent peers (18)[a]	.55 ± 1.0	.44 ± 0.8	.64 ± 1.0	.49 ± 1.0			

Note: $N = 922$ for every variable except parents' criminal conviction, where $N = 870$ (see text footnote 1).

[a]In the full cohort, the boys' mean score showed significantly worse level of risk on this variable than girls' mean score, all planned contrast $t(912) > 2.5$, all $p < .05$.

[b](a) LCP-path males scored significantly worse than AL-path males on 20 of the 26 risk measures, all planned contrast $t(912) > 1.9$, all $p < .05$; (b) AL-path females actually scored significantly better than AL-path males on 3 of the 26 risk measures, all planned contrast $t(912) > 2.5$, all $p < .05$; (c) AL-path females scored significantly worse than AL-path males on only 1 of the 26 risk measures, planned contrast $t(912) = 2.17$, $p < .05$; (d) LCP-path males scored significantly worse than AL-path females on 19 of the 26 risk measures, all planned contrast $t(912) > 1.7$, all $p < .05$.

Table 2. *Dunedin Study members on the life-course-persistent and adolescence-limited paths, compared on delinquent behavior and risk factors for delinquency*

	Comparison Groups' z Scores ($M \pm SD$)			LCP-AL Differences		
	Unclassified (N = 669; 73% of cohort)	Adolescence-Limited (N = 200; 22% of cohort)	Life-Course-Persistent (N = 53; 6% of cohort)	t Test	p	Effect Size in SD Units
Variety of delinquent acts (age in years)						
Males (15)	1.0 ± 1.4	7.3 ± 7.3	8.3 ± 7.0	1.24	*ns*	0.19
Males (18)	3.4 ± 2.0	11.1 ± 5.3	11.7 ± 6.2	0.92	*ns*	0.13
Females (15)	1.0 ± 1.5	8.4 ± 5.7	7.6 ± 2.1	0.70	*ns*	0.16
Females (18)	2.4 ± 2.9	7.3 ± 4.7	5.1 ± 5.1	1.91	.05	0.46
Parenting risk factors, z scored (age in years)						
Parents' criminal conviction	–0.03 ± 0.9	0.05 ± 1.0	0.20 ± 1.1	0.91	*ns*	0.15
Mother's age at her first birth	0.07 ± 1.0	–0.12 ± 1.0	–0.45 ± 0.9	2.06	.03	0.33
Mother–child observation (3)	–0.10 ± 0.9	0.02 ± 1.0	0.28 ± 1.0	1.76	.07	0.38
Harsh discipline (7–9)	–0.08 ± 0.9	0.11 ± 1.1	0.41 ± 0.9	2.00	.04	0.30
Inconsistent discipline (7–9)	–0.07 ± 0.9	0.10 ± 1.0	0.46 ± 1.2	2.38	.01	0.36
Moos family conflict (7–9)	–0.06 ± 1.0	0.14 ± 1.0	0.53 ± 1.0	2.43	.01	0.39
Mother's mental health (7–11)	–0.06 ± 1.0	–0.07 ± 0.9	0.58 ± 1.2	4.12	.01	0.51
Caregiver changes (birth–11)	–0.06 ± 0.9	0.04 ± 1.0	0.43 ± 1.2	2.57	.01	0.39
Years single parent (birth–11)	–0.02 ± 1.0	0.06 ± 1.0	0.34 ± 1.3	1.83	.06	0.28
Family SES (birth–15)	0.09 ± 1.0	–0.08 ± 1.0	–0.43 ± 0.9	2.36	.01	0.35
Child neurocognitive risk factors, z scored (age in years)						
Neurological abnormality (3)	–0.05 ± 0.9	–0.04 ± 0.9	0.24 ± 1.3	2.03	.04	0.28
Bayley motor test (3)	0.01 ± 1.0	0.10 ± 0.8	–0.13 ± 0.9	1.58	*ns*	0.23
Peabody Vocabulary (3)	0.00 ± 1.0	0.05 ± 1.0	–0.11 ± 0.9	1.09	*ns*	0.16
Binet IQ (5)	0.07 ± 0.9	0.05 ± 1.0	–0.31 ± 0.9	2.48	.01	0.36
WISC-R VIQ (7, 9, 11)	0.02 ± 1.0	0.09 ± 1.0	–0.31 ± 1.0	2.64	.00	0.40
Reading (7, 9, 11)	0.02 ± 1.0	0.11 ± 1.0	–0.46 ± 1.0	3.73	.01	0.57
Neuropsych memory (13)	0.04 ± 1.0	–0.02 ± 1.0	–0.41 ± 1.2	2.43	.01	0.39
Heart rate (7, 9, 11)	0.08 ± 1.0	–0.14 ± 0.9	–0.51 ± 0.9	2.44	.01	0.37
Child temperament-behavior risk factors, z scored (age in years)						
Difficult to manage (2)	–0.01 ± 1.0	0.00 ± 1.0	0.39 ± 0.8	2.61	.01	0.39
Under control observed (3)	–0.02 ± 1.0	–0.06 ± 0.9	0.45 ± 1.2	3.63	.01	0.51
Hyperactive, parent (5–11)	–0.08 ± 1.0	–0.11 ± 0.9	1.00 ± 1.1	7.38	.01	1.11
Hyperactive, teacher (5–11)	–0.12 ± 1.0	–0.07 ± 0.8	1.32 ± 1.1	9.50	.01	1.39
Fighting, parent (5–11)	–0.05 ± 1.0	0.02 ± 0.9	1.05 ± 0.9	7.11	.01	1.03
Fighting, teacher (5–11)	–0.10 ± 0.9	–0.09 ± 0.8	1.32 ± 1.2	10.03	.01	1.41
Peer rejection, parent (5–11)	–0.02 ± 0.9	–0.21 ± 0.7	0.95 ± 1.3	8.14	.01	1.16
Peer rejection, teacher (5–11)	–0.05 ± 1.0	–0.16 ± 0.8	0.91 ± 1.3	7.34	.01	1.07
Peer delinquency in adolescence, z scored (age in years)						
Delinquent peers (13)	–0.14 ± 1.0	0.34 ± 1.0	0.58 ± 1.2	1.47	*ns*	0.24
Delinquent peers (18)	–0.21 ± 0.9	0.58 ± 1.0	0.54 ± 1.0	0.29	*ns*	0.04

Note: ns, $\alpha > .10$. N = 922 for every variable (df = 919 for planned contrasts) except parents' criminal conviction, where N = 870 (see text footnote 1).

a small effect (SD = −.21), but the negative sign shows that children on the AL path were *less likely* than the average child to be disliked by their peers in primary school. In general, the AL-path group did not differ from the unclassified study members (mean effect

size: *SD* = .09). As expected, the unclassified study members did not differ from the normative standard on childhood risk (mean effect size: *SD* = .05).

Consistent with the hypothesis that young people on the AL path are aware of the delinquent behavior of their peers, the AL-path group scored .34 standard deviations higher than the norm on peer delinquency at age 13 years, and .58 standard deviations higher at age 18 years. The AL-path group had more delinquent peers than the unclassified group at both ages ($p < .001$). The LCP-path group also reported delinquent peers at ages 13 and 18 years. However, we and others have shown that although both childhood- and adolescent-onset youth say they have delinquent peers, when prior behavioral history is controlled peers' delinquency no longer predicts the delinquency of early-onset offenders but continues to predict the delinquency of late-onset offenders. This is true for males (Jeglum–Bartusch et al., 1997; Simons et al., 1994; Vitaro et al., 1997) and females (Caspi et al., 1993), and it is consistent with the taxonomic theory's hypothesis that LCPs attract delinquent peers during adolescence, whereas ALs are attracted to and influenced by delinquent peers (Moffitt, 1993).

Discussion

The childhood background of delinquents on the LCP path is pathological, but the background of delinquents on the AL path is normative

Whether we analyzed the Dunedin data separately for the sexes or together, the few study members on the LCP path fared poorly on background risk factors including poor parenting, neurocognitive risk, difficult temperament, and inattention–hyperactivity. Study members on the AL path, despite being involved in delinquency to the same extent as their counterparts on the LCP path, tended to have backgrounds that were normative or sometimes better than the average Dunedin child. These findings about differential childhood risk for childhood-onset versus adolescent-onset offenders are generally in keeping with findings reported from nine samples in six countries (Aguilar et al., 2000; Arseneault, Tremblay, Boulerice, & Saucier, 2000; Dean, Brame, & Piquero, 1996; Fergusson et al., 2000; Kratzer & Hodgins, 1999; Nagin et al., 1995; Patterson et al., 1998; Piquero, in press; Raine et al., 2000; Taylor et al., 2000; Tibbetts & Piquero, 1999). These studies include an unpredicted finding or two (e.g., an unanticipated trajectory group, low performance IQ for LCPs), but overall the taxonomic theory's prediction about differential childhood risk has survived the tests.

The differential-risk prediction encountered a particular challenge from a longitudinal study of a low-SES Minneapolis sample (Aguilar et al., 2000). This research team observed that differences between their childhood-onset and adolescent-onset groups were not significant for neurocognitive and temperament measures taken prior to age 3 years, but significant differences emerged only later in childhood. The authors inferred that neurocognitive and temperamental problems are later-emerging *consequences* of childhood psychosocial adversity but not early-emerging *contributing causes* for childhood-onset antisocial behavior. Their inference lead them to propose that psychosocial adversity is sufficient to account for initiation of the pathway into LCP antisocial behavior. Exclusive social hypotheses are probably not defensible, in view of emerging evidence that LCP-type antisocial behavior appears to have more heritable liability than AL antisocial behavior (DiLalla & Gottesman, 1989; Edelbrock et al., 1995; Lyons, True, Eisen, Goldberg, Meyer, Faraone, Eaves, & Tsuang, 1995; Taylor et al., 2000). Nonetheless, we are obliged to account for why our findings diverge from those of Aguilar et al.

The lack of significant early-childhood differences in the Minneapolis study may indicate flaws in the theory, or it may arise from methodological features such as the unrepresentative nature of the sample (homogeneous low SES, high risk), unusual sex composition of the groups (more males never antisocial, more females antisocial), weak psychometric qualities of infancy measures (i.e., predictive validity), or weak statistical power (only 35

adolescent-onset and 38 childhood-onset participants). Aguilar et al. concluded that perinatal problems and early cognitive measures were nonsignificant as predictors of the LCP path. Measures of perinatal complications would be expected to yield limited variation in the small, homogeneous sample, because pathological signs are quite rare in surviving infants and standardized measures such as the Bayley Scale at age 9 months are known for their poor predictive validity (McCall & Carriger, 1993). As a result, it is possible that the failure of these measures to predict the LCP path is part of the measures' more general failure to predict. Other studies have reported a significant relation between birth complications or low birth weight and LCP-type offending, but they used large representative cohorts, and the relation was seen primarily when the interaction between perinatal risk and socioeconomic status was tested (Arseneault et al., 2000; Arseneault et al., 2000; Kratzer & Hodgins, 1999; Tibbetts & Piquero, 1999; Raine et al., 1994), suggesting that such methodological features are needed to uncover subtle effects from the beginning of infancy.

The effect sizes reported here for the LCP–AL differences in cognitive risk, like those reported by Aguilar et al., increased with age, from small effects for neurological abnormalities, motor skills, and the Peabody vocabulary test at age 3 years, to medium effects for verbal IQ and reading at ages 7, 9, and 11 years. Our LCP–AL differences for behavioral risk also increased with age, from a small effect for difficult-to-manage at age 2 years to a medium effect for undercontrol observed at age 3 years, to a large effect for hyperactivity at ages 5–11 years. We suspect this progression of effect sizes is an artifact of a progression from infancy to childhood in the psychometric qualities of measurements (e.g., from single items to reliable aggregate scales, from rare pathognomic signs to normal distributions). In any case, we would not view this progression as problematic for the theory. The theory underscored the incremental construction of the antisocial personality. Therefore, it did not mention, but neither did it deny, the likely possibility that intellectual difficulties are also incrementally exacerbated when children endure long-term adversity. The theory noted that "discipline problems and academic failures *accumulate increasing momentum*" and "the life-course-persistent type has its origins in neuropsychological problems that *assume measurable influence* when difficult children interact with criminogenic home environments" (Moffitt, 1993, p. 695; italics added). If the progressive increase in effect sizes with age should be shown to be real, it is not inconsistent with the theory that LCP antisocials experience unique risk.

The developmental typology fits both sexes

Findings in this article suggest that the taxonomy describes parsimoniously the antisocial development of both males and females. Moreover, as predicted, the sex difference is very large for the LCP form of antisocial behavior (10 : 1 in this sample), whereas the sex difference is negligible for the AL form (1.5 : 1).

Only a few studies have tested the taxonomy while including sex comparisons, but it appears that our findings about females are broadly consistent with previous studies. Fergusson et al. (2000), studying the Christchurch sample (n = 1000), found that a single model described male and female trajectories of antisocial behavior, and the male to female ratio was 4 : 1 for early-onset subjects versus only 2 : 1 for late-onset subjects. Kratzer and Hodgins (1999), studying a Swedish cohort (n = 13000), found similar childhood risk factors for males and females in the LCP group, and the male to female ratio was 15 : 1 for early-onset subjects versus only 4 : 1 for late-onset subjects. Mazerolle et al. (2000), studying a Philadelphia cohort (n = 3655), reported that early onset signaled persistent and diverse offending for males and females alike. Tibbetts and Piquero (1999), studying a Philadelphia cohort (n = 987), found too few females on the LCP path to study their backgrounds with adequate statistical power. All studies concur that females are seldom childhood-onset or LCP-type (the exception is Aguilar et al., 2000, whose early-onset group had as many girls as boys).

A barrier to testing how our developmental taxonomy applies to the sexes is the difficulty

of gathering parent-, teacher- or self-reports to measure trajectories of antisocial behavior while simultaneously studying a sample large enough to ensure power for examining females. Low power apparently prevented some researchers who sampled both sexes from comparing them (Raine et al., 2000; Aguilar et al., 2000). Kratzer and Hodgins (1999) and Mazerolle et al. (2000) had samples large enough to compare the sexes, but consequently had to rely on official criminal conviction records to measure antisocial behavior. Conviction records are not optimal for the purpose of differentiating between childhood- and adolescent-onset comparison groups because children are seldom convicted, conviction taps only a fraction of even the most serious offenders, and people are on average not convicted until 4–5 years after they begin offending (Moffitt et al., 2001; Office of Juvenile Justice and Delinquency Prevention, 1998). Conviction data are more useful for defining continuously distributed features of LCP offending for study, such as diversity of offenses, violence, relatively early onset, and recidivistic persistence, but all of these are rare among females (Mazerolle et al., 2000; Tibbetts & Piquero, 1999). Despite these practical barriers to studying females, the data reported here, and other accumulating evidence, suggest two conclusions: first, the vast majority of female delinquents fit the AL-late-starter pattern; second, the childhood backgrounds of females who exhibit adolescent-onset antisocial behavior are normative, and certainly not pathological.[4]

We suggest that the theories of the origins of LCP and AL offending are explanatory across the sexes and irrespective of sex. According to one of the theories, LCP antisocial behavior emerges when inherited or acquired neurodevelopmental vulnerabilities are present in childhood and promote transactions with criminogenic environments. The rarity of females among LCP offenders makes sense because females as a group have been shown to experience lower levels than males of risk factors such as neurocognitive deficit, undercontrolled temperament, and hyperactivity (Earls, 1987; Eme, 1992; Moffitt et al., 2001). According to the other theory, AL antisocial behavior emerges in teenagers who do not have neurodevelopmental vulnerabilities, because these young people mimic antisocial peers in an effort to cope with their dysphoria in the maturity gap. The ubiquity of females among AL offenders follows from empirical observations that females are most antisocial soon after puberty, and when they are under the influence of relationships with males, who are more antisocial than females on average (Moffitt et al., 2001, review studies documenting these effects). In other words, no special female-specific theory is needed. How could Silverthorn and Frick (1999) have concluded that delinquent girls with adolescent onset have high-risk backgrounds? Their review drew on studies of community samples to correctly deduce that most girls' antisocial behavior onsets in adolescence (girls on the AL path would dominate community samples). However, they drew on studies of clinical and adjudicated samples for descriptions of the high-risk backgrounds of antisocial girls, incorrectly deducing that all antisocial girls have such backgrounds (girls on the LCP path, though rare in the population, probably dominate such highly selected samples). This illustrates that theory about the population is best built on data that represent the population.

More research is needed about girls and boys on the adolescent-onset path

Most research on the taxonomy to date has focused on testing hypotheses about the etiology of LCP offenders. Findings that this

4. A reviewer pointed out that if the six Dunedin girls on the LCP path had unusually early puberty they might still fit Silverthorne and Frick's view that all delinquent girls' onset of problems is postpubertal. However, the LCP girls' mean age of menarche was 13 years 1 month (SD = 17 months), which was 1 month older than the sample mean of 13 years, and 3 months older than the AL girls' mean of 12 years 9 months (SD = 12 months). This difference was not statistically significant, $t(393) = .51$, $p = .61$. Although menarche comes late in the pubertal process, Dunedin LCP girls' menarche occurred on average 6 years after the measure at age 7 years of problem behavior necessary to define the LCP's persistent behavior problems over three assessments (ages 7, 9, and 11 years). Thus, it is highly unlikely that LCP girls' puberty began before their behavior problems.

group can be distinguished in the early years of life have garnered much attention, contributing to the current enthusiasm for early-childhood interventions. Unfortunately, AL offenders have been relegated to the status of a contrast group and the original hypothesis about the distinct etiology of adolescent-onset offending has not captured the research imagination (but see Brezina, 2000; Bukowski et al., 2000; Zebrowitz et al., 1998). This is unfortunate because adolescent-onset offenders are quite common (one quarter of both males and females, as defined in this study), and they are not benign.

Aguilar et al. (2000) discovered that adolescent-onset youths experienced elevated internalizing symptoms and perceptions of stress at age 16 years, bringing a fresh emphasis to the taxonomy's assertion that these adolescents experience dysphoria. Fergusson et al. (2000) pointed out that a normative developmental history is not necessarily a salutary developmental history, and that the normative, moderate levels of risk in the backgrounds of AL youngsters may leave them without protection against delinquent peer influence. Kratzer and Hodgins (1999) drew attention to the risk of criminal conviction in adulthood for people with late-onset offending. We have shown that the antisocial behavior of AL offenders is not inconsequential; in fact, it exposes them to numerous snares during the adolescent years (e.g., leaving school without credentials, becoming a teen parent, developing dependency on tobacco, drugs or alcohol, serious injury, sexually transmitted diseases, a criminal record, and incarceration; Moffitt et al., 1996, 2001). Such snares can compromise their ability to make a successful transition to adulthood, impair their health, and set in motion a snowball of cumulative disadvantage (Moffitt, 1993). Almost all females who engage in antisocial behavior fit the AL path, and their adult outcomes can be very poor (Moffitt et al., 2001; Robins, 1986).

The theory of AL antisocial behavior regards it as an adaptation response to modern teens' social context, not the product of a cumulative history of pathological maldevelopment. Nonetheless, it specifies that AL behavior often attracts harmful consequences, and it does not presume that the problems of AL offenders will remit spontaneously without remediation efforts (Moffitt et al., 1996). Legal scholars point out that harsh sentencing applied to AL delinquents incurs societal costs, including damaged future employment prospects and delayed desistence from crime. These scholars call for juvenile justice policy that gives AL delinquents "room to reform" (Scott & Grisso, 1997, p. 180). This article and our previous report (Moffitt et al., 1996) have shown that young people on the AL path lack a pathological history, problem personalities, low IQ, reading failure, inadequate parents, and broken attachment relationships, suggesting that they should be ideal candidates for intervention. Therefore, we hope this article will stimulate more research to improve knowledge about the AL developmental path.

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Appendix: Twenty-six Measures of Childhood Risk and Two Measures of Peer Delinquency

Measures were selected from the Dunedin study archives to tap the three risk domains of family adversity and inadequate parenting, child neurocognitive health, and child temperament and behavior. Measures are described below; sex differences are reported in Table 1.

Family adversity and inadequate parenting risk predictors

Parental criminality was assessed via a questionnaire posted to mothers and fathers when the study members were young adults and the parents' ages ranged from 40 to 75 years. The parents were asked if they had ever been convicted of a crime. At least one parent responded for 91% of the study members; 13% of these had at least one parent who reported conviction for crime (Moffitt et al., 2001). Higher score indicated more risk.

Mother's age at her first birth measured whether the study member's mother had been a teenage parent, regardless of her age when the study child was born (Jaffee, Moffitt, Caspi, Belsky, & Silva, 2001). Lower score indicated more risk.

Deviant mother–child interaction was assessed at age 3 years when the mother was observed during a 1-hr testing session and rated by an observer on eight categories. A point was assigned for each category on which the interaction appeared negative or inappropriate (e.g., if the mother's evaluation of the child was constantly critical or derogatory, or if she was rough or inconsiderate in physically handling the child) and the points were summed. Details for this scale in the Dunedin study are described by Henry, Caspi, Moffitt, and Silva (1996). Higher score indicated more risk.

Harsh discipline was measured at ages 7 and 9 years using a checklist of disciplinary behaviors on which parents indicated if they engaged in 10 behaviors, such as "smack [your child] or hit him/her with something," "try to frighten [your child] with someone like his/her father or a policeman," and "threaten to smack, or deprive [your child] of something." Details for this scale in the Dunedin study are described by Magdol, Moffitt, Caspi, and Silva (1998). Higher score indicated more risk.

Inconsistent discipline was measured at ages 7 and 9 years as part of an interview about how parents dealt with the study child when he or she was naughty or misbehaved. Mothers evaluated their own discipline, as well as their husbands' discipline, on a 4-point scale (1, *always the same;* 4, *very changeable*) and the responses were summed (details in Moffitt et al., 2001). Higher score indicated more risk.

Family conflict was measured at ages 7 and 9 years with the Moos Family Relations Index (Moos & Moos, 1981), completed by the mothers. The conflict subscale contains items such as "Family members sometimes hit each other." Details for this scale in the Dunedin study are described by Parnicky, Williams, and Silva (1985). Higher score indicated more risk.

Mother's mental health problems were measured with the Malaise Inventory completed by the mothers when the study members were ages 7, 9, and 11 years. The 24-item questionnaire assesses a variety of common symptoms reflecting affective stress response (e.g., easily upset, miserable) and somatic symptoms (e.g., tiredness, headaches; Rodgers, Pickles, Power, Collishaw, & Maughan, 1999). Details about this scale in the Dunedin study are described by McGee, Williams, and Silva (1985a). Higher score indicated more risk.

Number of caregiver changes experienced by the child was assessed from birth through age 11 years (range 0–6). At each assessment year the parents were asked what changes in the family configuration had occurred since the last assessment. Responses included changes such as parent death, separation, cohabitation, remarriage, child sent to relatives, or foster care. Higher score indicated more risk.

Years with a single parent indexes the number of years from birth to age 11 years that the study member lived with a single parent. Details about these last two measures are described by Henry, Moffitt, Robins, Earls, and Silva (1993). Higher score indicated more risk.

Family socioeconomic status (SES) measured the average SES level of the study members' families across the first 15 years of the Dunedin study using a 6-point scale (1, unskilled laborer; 6, professional) designed for New Zealand (Elley & Irving, 1976). Details about SES measurement in the Dunedin study are described by Wright, Caspi, Moffitt, Miech, and Silva (1999). Lower score indicated more risk.

Child neurocognitive risk predictors

Neurological abnormalities were assessed at age 3 years when each child was examined by a pediatric

neurologist for neurological signs, including assessment of motility, passive movements, reflexes, facial musculature, strabismus, nystagmus, foot posture, and gait. This assessment followed procedures described by Touwen and Prechtl (1970); results for the Dunedin study are reported by McGee, Silva, and Williams (1984). Higher score indicated more risk.

Motor development was assessed at age 3 years by a pediatrician using the 28 most difficult items from the Bayley Scales of Infant Development (Bayley, 1969). Details about the Bayley in the Dunedin study have been reported by Silva and Ross (1980). Lower score indicated more risk.

Intelligence was assessed at age 3 years with the Peabody Picture Vocabulary Test (Dunn, 1965), at age 5 years with the Stanford Binet Intelligence Scales (Terman & Merrill, 1960), and at ages 7, 9, and 11 years, with the Wechsler Intelligence Scale for Children–Revised (WISC-R; Wechsler, 1974). We averaged the WISC-R Verbal IQ scores from the three age periods. All tests were administered by psychometrists according to standard protocol. Details about intelligence testing in the Dunedin study are described by Moffitt, Caspi, Harkness, and Silva (1993). Lower scores indicated more risk.

Reading achievement was measured at ages 7, 9, and 11 years by the Burt Word Reading Test (Scottish Council for Research in Education, 1976), a word-recognition reading test normed for New Zealand children, which resembles the American Wide-Range Achievement Test of reading. Details about this measure in the Dunedin study are described by Fergusson, Horwood, Caspi, Moffitt, and Silva (1996). We combined the (age-standardized) reading scores from the three age periods to form an overall score. Lower score indicated more risk.

Heart rate was measured by nurses at ages 7, 9, and 11 years. This measure is included as an indicator of the autonomic actions of the nervous system, as it has been linked to features of LCP antisocial behavior such as violence (Farrington, 1997). At each age, an average heart rate measure was derived from measures of resting heart rate taken on three occasions during the course of a physical examination. We combined the (age-standardized) measures of resting heart rate from the three age periods to form an overall score (details in Moffitt et al., 2001). Lower score indicated more risk.

Neuropsychological memory scores at age 13 years were constructed as a composite factor score from scores on the several trials of the Rey Auditory Verbal Learning Test, measuring immediate and delayed recall of word lists. Details about this measure are provided by Frost, Moffitt, and McGee (1989). Lower score indicated more risk.

Child temperament and behavioral risk predictors

Difficult temperament was assessed in two ways, by parental reports and by observations made by psychological examiners. At the child's third birthday mothers were asked whether the child had been easy or difficult to manage in the prior year. Response options coded were "easy all the time," "easy most of the time," or "very difficult." At age 3 years the children participated in a 90-min testing session of cognitive and motor tasks administered by an examiner who had no knowledge of the child's prior behavioral history. Following the session, the examiner rated the child's behavior on a checklist, and based on factor analyses of these ratings we identified a developmentally robust dimension reflecting individual differences in undercontrol. Children scoring high on this factor were emotionally labile, impulsive, irritable, negativistic, rough, and had difficulty concentrating. Details are presented by Caspi, Henry, McGee, Moffitt, and Silva (1995). Higher scores indicated more risk.

Hyperactivity was measured with the Rutter Child Scales (Rutter, Tizard, & Whitmore, 1970), supplemented with items concerning inattention, impulsivity, and hyperactivity from the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1980) criteria for attention deficit disorder. The scales were completed by parents and teachers at ages 5, 7, 9, and 11 years (see McGee, Williams, & Silva, 1985b, for details). The hyperactivity items were summed across the four age periods to derive scales, separately for parents and teachers. Higher scores indicated more risk.

Fighting was measured at ages 5, 7, 9, and 11 years. Parents and teachers reported whether the study child "frequently fights with other children," by rating 0 (*does not apply*), 1 (*applies somewhat*), or 2 (*certainly applies*). We summed these ratings across the four age periods to create scales, separately for parents and teachers (details in Moffitt et al., 2001). Highers score indicated more risk.

Peer rejection was measured at ages 5, 7, 9, and 11 years, when parents and teachers evaluated whether the child was "not much liked by other children" using the above-mentioned 3-point scale. We summed these ratings across the four age peri-

ods to create scales, separately for parents and teachers (details in Moffitt et al., 2001). Higher scores indicated more risk.

Peers' delinquency

Peer delinquency, assessed at age 13 years, is the number of norm violations and illegal offenses on the early delinquency scale which the study members had reported for "my friends and other people my age who I know." Details have been described by Caspi et al. (1993), who presented analyses showing that many study members' awareness of their peers' delinquency antedated onset of their own delinquency, demonstrating that study members' reports on this measure of peer delinquency are not merely a reflection of the reporters' own delinquency. Peer delinquency was not assessed at age 15 years. At age 18 years, study members reported the delinquency of their friends and the people they like to spend time with, using the items of the Self-reported Delinquency interview, as described by Moffitt et al. (1994). Higher scores indicated more risk.