CHAPTER 15

Life-Course-Persistent versus Adolescence-Limited Antisocial Behavior

TERRIE E. MOFFITT

A BRIEF INTRODUCTION TO THE TWO PROTOTYPES 571

THE HYPOTHESIS THAT LIFE-COURSE-PERSISTENT ANTISOCIAL DEVELOPMENT EMERGES FROM EARLY NEURODEVELOPMENTAL AND FAMILY ADVERSITY RISK FACTORS 572

What Research Is Needed? 574

THE HYPOTHESIS THAT GENETIC ETIOLOGICAL PROCESSES CONTRIBUTE MORE TO LIFE-COURSE-PERSISTENT THAN ADOLESCENCE-LIMITED ANTISOCIAL DEVELOPMENT 574

What Research Is Needed? 576

IS A THIRD GROUP NEEDED? CHILDHOOD-LIMITED AGGRESSIVE CHILDREN MAY BECOME LOW-LEVEL CHRONIC CRIMINAL OFFENDERS WITH PERSONALITY DISORDERS 577

What Research Is Needed? 578

IS A FOURTH GROUP NEEDED? ADULT-ONSET ANTISOCIAL BEHAVIOR 578

THE HYPOTHESIS THAT ADOLESCENCE-LIMITED ANTISOCIAL BEHAVIOR IS INFLUENCED BY THE MATURITY GAP AND BY SOCIAL MIMICRY OF ANTISOCIAL MODELS 579

What Research Is Needed? 581

THE HYPOTHESIS THAT ABSTAINERS FROM DELINQUENCY ARE RARE INDIVIDUALS WHO ARE EXCLUDED FROM NORMATIVE PEER GROUP ACTIVITIES IN ADOLESCENCE 581

What Research Is Needed? 582

THE HYPOTHESIS THAT LIFE-COURSE-PERSISTENT AND ADOLESCENCE-LIMITED DELINQUENTS DEVELOP DIFFERENT PERSONALITY STRUCTURES 582

What Research Is Needed? 583

THE HYPOTHESIS THAT LIFE-COURSE-PERSISTENT DEVELOPMENT IS DIFFERENTIALLY ASSOCIATED IN ADULTHOOD WITH SERIOUS OFFENDING AND VIOLENCE 583

What Research Is Needed? 584

THE HYPOTHESIS THAT CHILDHOOD-ONSET ANTISOCIAL BEHAVIOR WILL PERSIST INTO MIDDLE ADULTHOOD, WHEREAS ADOLESCENT-ONSET ANTISOCIAL BEHAVIOR WILL DESIST IN YOUNG ADULTHOOD 585

What Research Is Needed? 588

GENDER: THE HYPOTHESIS THAT MOST FEMALES ANTISOCIAL BEHAVIOR IS THE ADOLESCENCE-LIMITED TYPE 588

What Research Is Needed? 590

RACE: THE HYPOTHESIS THAT BOTH LIFE-COURSE-PERSISTENT AND ADOLESCENCE-LIMITED DEVELOPMENTAL PROCESSES ARE EXACERBATED BY SOCIETAL RACE PREJUDICE 590

What Research Is Needed? 591

RESEARCH NEEDED ON OTHER HYPOTHESES 591

CONCLUSIONS 592

REFERENCES 593

This chapter reviews 10 years of research into a developmental taxonomy of antisocial behavior that proposed two primary hypothetical prototypes: life-course-persistent versus adolescence-limited offenders. The taxonomic theory was fully articulated for the first time in a chapter written for the first edition of Developmental Psychopathology (Caspì & Moffitt, 1995). Therefore, it is particularly appropriate to review for the second edition the research published since then. According to the taxonomic theory, life-course-persistent offenders' antisocial behavior has its origins in neurodevelopmental processes; it begins in childhood and continues persistently thereafter. In contrast, adolescence-limited offenders' antisocial behavior has its origins in social processes; it begins in adolescence and desists in young adulthood. According to the theory, life-

Discussions in the literature have pointed out that if the taxonomic theory is proven accurate, it could usefully improve classification of subject groups for research (Nagin, Farrington, & Moffitt, 1995; Silverthorn & Frick, 1999; Zucker, Ellis, Fitzgerald, Bingham, & Sanford, 1996), focus research into antisocial personality and violence toward the most promising causal variables (Brezina, 2000; Lahey, Waldman, & McBurnett, 1999; Laucht, 2001; Osgood, 1998), and guide the timing and strategies of interventions for delinquent types (Howell & Hawkins, 1998; Scott & Grissos, 1997; Vermeiren, 2002). Several writers have extracted implications for intervention from the taxonomy. Howell and Hawkins observed that preventing life-course-persistent versus adolescence-limited antisocial behavior requires interventions that differ in both timing and target. Preventing life-course-persistent lifestyles requires early childhood interventions in the family. In contrast, adolescence-limited offending ought to be prevented by treating adolescents individually to counteract peer influence (instead of in groups that facilitate deviant peer influence; Dishion, McCord, & Poulin, 1999). Scott and Grissos argued compellingly that the juvenile justice system should identify adolescence-limited delinquents and give them room to reform. Surveys of juvenile court judges and forensic psychologists reveal that the offender characteristics they rely on to recommend a juvenile for transfer to adult court match the characteristics that distinguish life-course-persistent delinquents (Saakkin, Yff, Neumann, Leistico, & Zalot, 2002). In contrast, Scott and Grissos argue that sending life-course-persistent delinquents to adult court is inappropriate because the cognitive deficits typical of these delinquents render them unlikely to meet legal criteria for competency to stand trial.


The reader is referred to two prior publications that articulate the main hypotheses derived from this taxonomic theory. The first article published that proposed the two prototypes and their different etiologies ended with a section headed “Strategies for Research,” which described predictions about epidemiology, age, social class, risk correlates, offense types, desistance from crime, abstainers from crime, and the longitudinal stability of antisocial behavior (Moffitt, 1993, pp. 694–696). The article specified which findings would disconfirm the theory. A version published elsewhere specified disconfirmable hypotheses about sex and race (Moffitt, 1994). When these hypotheses from the taxonomy were put forward 10 years ago, none of them had been tested, but since then several have been tested by us and by others. This chapter reviews the results of that research, as of summer 2004, and points out where more research is needed.

A BRIEF INTRODUCTION TO THE TWO PROTOTYPES

In a nutshell, we suggested that life-course-persistent antisocial behavior originates early in life, when the difficult behavior of a high-risk young child is exacerbated by a high-risk social environment. According to the theory, 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. Opportunities to learn prosocial skills are lost. Over the first 2 decades of development, transactions between the individual and the environment gradually construct a disordered personality with hallmark features of physical aggression and antisocial behavior persisting to midlife. The theory predicts that antisocial behavior will infiltrate multiple adult life domains: illegal activities, problems with employment, and victimization of intimate partners and children. This infiltration diminishes the possibility of reform.

In contrast, we suggested that adolescence-limited antisocial behavior emerges alongside puberty, when otherwise ordinary healthy youngsters experience psychological discomfort during the relatively role-less years between their biological maturation and their access to mature privileges and responsibilities, a period we called the “maturity gap.” They experience dissatisfaction with their dependent status as a child and impatience for what they anticipate are the privileges and rights of adulthood. While young people are in this gap, it is virtually normative for them to find
the delinquent style appealing and to mimic it as a way to demonstrate autonomy from parents, win affiliation with peers, and hasten social maturation. However, because their predelinquent development was normal, most adolescence-limited delinquents are able to desist from crime when they age into real adult roles, returning gradually to a more conventional lifestyle. This recovery may be delayed if the antisocial activities of adolescence-limited delinquents attract factors we called “snares,” such as a criminal record, incarceration, addiction, or truncated education without credentials. Such snares can compromise the ability to make a successful transition to adulthood.

The literature contains other theoretical statements about early- versus late-onset antisocial behavior, but our theory differed in three ways. First, it offered not only an account of onset processes but also included an explanation of the developmental processes leading to the maintenance of and desistance from antisocial behavior. Second, whereas other theories emphasize inept parenting as the primary cause initiating life-course-persistent antisocial behavior, this theory argued that children’s own characteristics are a primary force in the transactions between child and environment. Third, whereas other theories emphasize poor parental supervision and monitoring as the primary cause of adolescence-limited delinquent behavior, this theory argued that adolescent-limited delinquency emerges from the age-appropriate developmental process of building autonomy, in which young people move away from childhood parent-child relationships and toward mature peer-to-peer relationships.

THE HYPOTHESIS THAT LIFE-COURSE-PERSISTENT ANTISOCIAL DEVELOPMENT EMERGES FROM EARLY NEURODEVELOPMENTAL AND FAMILY ADVERSITY RISK FACTORS

The original hypothesis about childhood risk specified that predictors of life-course-persistent antisocial behavior should 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, but not age” (Moffitt, 1993, p. 695).

Our own tests of this hypothesis have been carried out in the Dunedin Multidisciplinary Health and Development Study, a 32-year longitudinal study of a birth cohort of 1,000 New Zealanders. A full description of the Dunedin Study and the New Zealand research setting can be found in Moffitt, Caspi, Rutter, and Silva (2001). These tests have examined childhood predictors measured between ages 3 and 13, operationalizing the two prototypes of antisocial behavior using both categorical and continuous statistical approaches. These studies showed that the life-course-persistent path was differentially predicted by individual risk characteristics, including undercontrolled temperament measured by observers at age 3, neurological abnormalities and delayed motor development at age 3, low intellectual ability, reading difficulties, poor scores on neuropsychological tests of memory, hyperactivity, and slow heart rate (Jeglum-Bartusch, Lynam, Moffitt, & Silva, 1997; Moffitt, 1990; Moffitt & Caspi, 2001; Moffitt, Lynam, & Silva, 1994). The life-course-persistent path was also differentially predicted by parenting risk factors, including teenage single parent, mother with poor mental health, and mother who was observed to be harsh or neglectful, as well as by experiences of harsh and inconsistent discipline, much family conflict, many changes of primary caretaker, low family socioeconomic status (SES), and rejection by peers in school. In contrast, study members on the adolescence-limited path, despite being involved in teen delinquency to the same extent as their counterparts on the life-course-persistent path, tended to have backgrounds that were normative, or sometimes even better than the average Dunedin child’s (Moffitt & Caspi, 2001). A replication of this pattern of differential findings was reported by a study of 800 children followed from birth to age 15 years (Brennan, Hall, Bor, Najman, & Williams, 2003). An early-onset persistent antisocial group, an adolescent-onset antisocial group, and a nonantisocial group were identified. Measured biological risks (e.g., neuropsychological test deficits at age 15) and childhood social risks (e.g., harsh discipline, maternal hostility), and an interaction between these two risks predicted membership in the early-onset persistent group, but membership in the adolescent-onset group was unrelated to childhood social risks or biological risks.

The Dunedin findings about differential neurodevelopmental and family risk correlates for childhood-onset versus adolescent-onset offenders are generally in keeping with findings reported from other samples in Australia, Canada, England, Mauritius, New Zealand, Norway, Russia, Sweden, and several states in the United States. These studies operationalized the types using a variety of conceptual approaches, many different measures of antisocial behaviors, and very different statistical methods (Aguilar, Sroufe, Egeland, & Carlson, 2000; Arseneault, Tremblay, Boulerice, & Saucier, 2002; Brennan et al., 2003; Chung, Hill, Hawkins, Gilchrist, & Nagin, 2002; Dean, Brame, & Piquero, 1996; Donnellan, Ge, & Wenk, 2000; Fergusson,
Horwood, & Nagin, 2000; Kjelsberg, 1999; Kratzer & Hodgins, 1999; Lahey et al., 1998; Magnusson, af Klintberg, & Stattin, 1994; Maughan, Pickles, Rowe, Costello, & Angold, 2001; Mazzerolle, Brame, Paternoster, Piquero, & Dean, 2000; McCabe, Hough, Wood, & Yeh, 2001; Nagin et al., 1995; Nagin & Tremblay, 1999, 2001b; Patterson, Forgatch, Yoerger, & Stoolmiller, 1998; Piquero, 2001; Piquero & Brezina, 2001; Raine et al., 2005; Raine, Yaralian, Reynolds, Venables, & Mednick, 2002; Roeder, Lynch, & Nagin, 1999; Ruchkin, Koposov, Vermeiren, & Schwab-Stone, 2003; Tibbetts & Piquero, 1999; Tolan & Thomas, 1995; Wiesner & Capaldi, 2003). Each of these studies added support for the taxonomy’s construct validity by reporting differential correlates for early-onset/persistent antisocial behavior versus later-onset/temporary antisocial behavior. However, at least one research team found mixed evidence for the taxonomy (cf. Brame, Bushway, & Paternoster, 1999, versus Paternoster & Brame, 1997).

Other studies, although not necessarily presented as a formal test of the two types, have reported findings consonant with our predictions about the types’ differential childhood risks. For example, children’s hyperactivity interacts with poor parenting skills to predict antisocial behavior that has an early onset and escalates to delinquency (Patterson, DeGarmo, & Knutson, 2000), an interaction that fits the hypothesized origins of the life-course-persistent path. Other studies have reported that measures reflecting infant nervous system maldevelopment interact with poor parenting and social adversity to predict aggression that is chronic from childhood to adolescence (Arseneault et al., 2002). Measures indexing infant nervous system maldevelopment and social adversity also interact to predict early-onset violent crime (Raine, Brennan, & Mednick, 1994; Raine, Brennan, Mednick, & Mednick, 1996) but do not predict nonviolent crime (Arseneault, Tremblay, Boulerice, Seguin, & Saucier, 2000; Raine, Brennan, & Mednick, 1997). Two additional findings are consistent with our prediction that infant nervous system maldevelopment contributes to long-term life-course-persistent antisocial outcomes. First, prenatal malnutrition has been found to predict adult Antisocial Personality Disorder (Neugebauer, Hoek, & Susser, 1999). Second, adults with Antisocial Personality Disorder exhibit two nervous system abnormalities attributable to disruption of brain development in early life: enlargement of the corpus callosum, assessed by structural magnetic resonance imaging, and abnormal corpus callosum connective function, assessed by divided visual field tests (Raine et al., 2003).

Our 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 neuropsychological and temperament measures taken prior to age 3, although they found that significant differences did emerge later in childhood. The authors inferred that childhood psychosocial adversity is sufficient to account for the origins of life-course-persistent antisocial behavior, which is similar to Patterson and Yoerger’s (1997) thesis that unskilled parenting is sufficient to account for the early-onset antisocial type. Such exclusive socialization hypotheses are probably not defensible in view of emerging evidence that the life-course-persistent pattern of antisocial behavior appears to have substantial heritable liability (DiLalla & Gottesman, 1989; Eley, Lichtenstein, & Moffitt, 2003; Taylor, Iacono, & McGue, 2000), a finding we revisit later in this chapter. The lack of significant early childhood differences in the Minneapolis study may have arisen from methodological features of the study, including the unrepresentative and homogeneous nature of the sample (all high-risk, low-SES families) and irregular sex composition of the groups (more females than males were antisocial), or weak psychometric qualities of the infant measures (unknown predictive validity). Infant measures are known for their poor predictive validity (McCall & Carriger, 1993), and thus it is possible that the failure of the infant measures to predict the life-course-persistent path is part of such measures’ more general failure to predict outcomes.

One study has reported that difficult temperament assessed at age 5 months distinguished a group of children who showed a trajectory of high rates of physical aggression, as compared to cohort peers, across ages 17, 30, and 42 months (Tremblay et al., 2004). However, until this cohort of 572 infants is followed beyond age 3.5 years into adolescence, we cannot be confident that they represent youngsters on the life-course-persistent pathway. Other studies have reported a significant relation between life-course-persistent-type offending and problems known to be associated with neuropsychological and temperament difficulties in infancy: perinatal complications, minor physical anomalies, and low birthweight (Arseneault et al., 2000, 2002; Kratzer & Hodgins, 1999; Raine et al., 1994; Tibbetts & Piquero, 1999). These studies illustrate desirable features for testing neurodevelopmental risks from the beginning of infancy for persistent antisocial behavior: large samples, representative samples, infant measures with proven predictive validity, and attention to interactions.
between neurodevelopmental and social adversity (Cicchetti & Walker, 2003).

**What Research Is Needed?**

Research already documents that life-course-persistent antisocial behavior has the predicted neurodevelopmental correlates in the perinatal and middle childhood periods, but the Aguilar et al. (2000) study remains the only one that has reported objective measures of infants' temperament and neurocognitive status prior to age 3 years, and it did not find the associations predicted by the theory. This study constitutes an important challenge that must be taken seriously, particularly as Brennan et al. (2003) also found no significant connection between temperament or vocabulary assessed in early life and early-onset persistent aggression. Clearly, more research is needed to fill in the critical gap between birth and age 3 years. This might be accomplished by following up the antisocial outcomes of infants tested with newer neurocognitive measures having documented predictive validity, such as the infant attention-habitation paradigm (Sigman, Cohen, & Beckwith, 1997).

Another feature of life-course-persistent theory that needs testing is the argument that antisocial behavior becomes persistent because a child's early difficult behavior provokes harsh treatment or rejection from parents, teachers, and peers, which in turn promotes more difficult child behavior. Adoption and twin studies have documented an initial "child effect"; that is, children carrying a genetic liability to antisocial behavior provoke harsh responses from their parents (Ge et al., 1996; Jaffee, Caspi, Moffitt, Polotomas, et al., 2004; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998; Riggins-Caspers, Cadoret, Knutson, & Langbehn, 2003). Such genetically informative studies should be followed up to ascertain whether this process, beginning with a child effect, ultimately leads to antisocial behavior that persists long term.

**THE HYPOTHESIS THAT GENETIC ETIOLOGICAL PROCESSES CONTRIBUTE MORE TO LIFE-COURSE-PERSISTENT THAN ADOLESCENCE-LIMITED ANTISOCIAL DEVELOPMENT**

Journalists have drawn the public's attention to families that appear to contain far more than their share of criminal family members for several generations (Butterfield, 1996, 2002). This concentration of crime in families has been confirmed by studies of large populations of families. In general, fewer than 10% of the families in any community account for more than half of criminal offenses (Farrington, Barnes, & Lambert, 1996; Farrington, Jolliffe, Loeber, Stouthamer-Loeber, & Kalb, 2001; Rowe & Farrington, 1997). Moreover, research has shown that parental crime and parental psychopathology are more strongly associated with early-onset antisocial behavior than late-onset antisocial behavior among offspring (Moffitt & Caspi, 2001; Raine et al., 2005; Taylor et al., 2000). These findings seem to implicate a genetic influence on life-course-persistent antisocial behavior, but they could also be explained by social transmission of antisocial behavior within families. Therefore, we next review studies using genetically sensitive research designs to glean insights about life-course-persistent antisocial behavior. If genetic etiological processes contribute more to life-course-persistent than to adolescence-limited antisocial development, we would expect to find that estimates of genetic influence are larger for antisocial behaviors committed by young children and adults than for antisocial behaviors committed by adolescents.

DiLalla and Gottesman (1989) first observed that adult crime seemed to be more heritable than adolescent juvenile delinquency. Our 1993 paper (Moffitt, 1993, p. 694) agreed with these authors that if the life-course-persistent type's causal factors are partly inherited, and if most antisocial adults are life-course-persistent but most antisocial adolescents are not, then this could account for the observed greater genetic influence on individual differences in adult than adolescent samples. As it turns out, the lack of heritability among juveniles in the DiLalla and Gottesman review probably resulted from low power and insensitive measurement; in 1989, the entire literature of behavior genetic studies of juvenile delinquency consisted of fewer than 200 twin pairs, and the measure of antisocial behavior was conviction, a rare outcome for juveniles. Since then, a large number of better-designed behavioral genetic studies have proven that juvenile antisocial behavior is at least somewhat heritable. However, among these, four groups of studies provide circumstantial evidence that life-course-persistent antisocial behavior does have stronger heritable origins than adolescence-limited antisocial behavior.

The first group comprises four studies of large representative samples of very young twins. Because life-course-persistent antisocial behavior begins early in life, if it is genetically influenced we would expect high heritability coefficients from studies of very young children. Dionne, Tremblay, Boivin, Laplante, and Perusse (2003) report 58% heritability for aggression among Canadian 19-month-olds. Van den Oord, Verhulst, and Boomsma (1996) report 69%
heritability for aggression among Dutch 3-year-olds. In a different Dutch cohort, van der Valk, Verhulst, Stroet, and Boomsma (1998) report 50% heritability for externalizing behaviors among 2- to 3-year-old boys and 75% for girls. Arseneault et al. (2003) report heritabilities of 61%, 69%, and 76% among British 5-year-olds for ratings of antisocial behaviors made by observers, mothers, and teachers, respectively. These high estimates for very young twins are in contrast to the lower estimate of 41% heritability from a meta-analysis of older samples (Rhee & Waldman, 2002).

A second group of studies has identified the two subtypes on the basis of the heterogeneity in the phenotype, often using the Aggression and Delinquency narrow-band scales from the Child Behavior Checklist (CBCL; Achenbach, 1985). The Aggression scale is thought to be associated with the life-course-persistent prototype because it measures antisocial personality and physical violence and its scores are stable across development, whereas the Delinquency scale is associated with the adolescence-limited prototype because it measures rule breaking and its mean scores rise steeply during adolescence (Stanger, Achenbach, & Verhulst, 1997). In fact, both life-course-persistent and adolescence-limited young people engage in the behaviors on the Delinquency scale, but adolescence-limited young people are relatively more numerous, and if they have less genetic risk, then we would expect the Delinquency scale to yield lower heritability estimates than the Aggression scale. Twin and adoption studies of these scales report higher heritability for Aggression (around 60%) than Delinquency (around 30% to 40%), but the shared environment is significant only for the Delinquency scale (also around 30% to 40%; e.g., Deater-Deckard & Plomin, 1999; Edelbrock, Rende, Plomin, & Thompson, 1995; Eley, Lichtenstein, & Stevenson, 1999; but see Schmitt, Fulker, & Mrazek, 1995). The approach of contrasting two phenotypes within antisocial behavior was also taken by Viding and colleagues (Viding, Blair, Moffitt, & Plomin, 2005), who reported that genes influenced 81% of the variation in antisocial behavior among callous-unemotional children, but only 30% of variation among the other study children, who engaged in ordinary antisocial behaviors. A different approach to contrasting two heterogeneous phenotypes was followed by Arseneault et al. (2003), who found that antisocial behavior that was pervasive across settings was more heritable than antisocial behavior that was situational; heritability was 82% if a child’s antisocial behavior was agreed on by four different reporters across settings at home and at school, but lower (28% to 51%) for antisocial behavior limited to one setting or one reporter.

A third group of studies has defined life-course-persistent antisocial behavior in terms of preadolescent onset, contrasting it against antisocial behavior that begins during the adolescent period. One study found early onset to be strongly familial and substantially heritable, in contrast to adolescent onset which was less familial and largely influenced by environment (Taylor et al., 2000). In a Swedish twin study, 5-year continuity from childhood to adolescence in the CBCL Aggression scale was largely mediated by genetic influences, whereas continuity in the Delinquency scale was mediated both by the shared environment and genetic influences (Eley et al., 2003).

A fourth group of studies has taken a developmental approach to the other end of the life span, defining life-course-persistent antisocial behavior in terms of presence in adolescence combined with subsequent persistence to adulthood Antisocial Personality Disorder. Two studies demonstrated that such persistent antisocial behavior was significantly more heritable than that limited to adolescence (Jacobson, Neale, Prescott, & Kendler, 2003; Lyons et al., 1995). These longitudinal studies are supported by a meta-analysis containing adolescent and adult samples assessed with similar measures of aggression, in which adult samples generated significantly higher heritability estimates, on average, than adolescent samples (Miles & Carey, 1997). Rhee and Waldman’s (2002) meta-analysis did not find higher heritability for adults than adolescents, because in the pool of studies they examined, age was wholly confounded with reporting source; adolescent studies used rating scales, and adult studies used official crime records.

Taken together, the four groups of existing studies suggest that the pattern of antisocial behavior that (1) begins early in life, (2) is pervasive across settings, (3) is characterized by aggressive personality traits, (4) includes physical aggression, and (5) persists into adulthood is associated with relatively more genetic influence than is the pattern of later-onset, situational, transient delinquency. What does this high heritability estimate mean? It does not mean that environmental experiences have a negligible effect on life-course-persistent antisocial development. To the contrary, a heritability estimate, as calculated, reflects the additive effects of specific genes on a phenotype, but it also includes two types of interplay between genes and environments. Correlations between genes and environments (GrE) increase the heritability estimate if genes lead study members to encounter environments that in turn exacerbate the phenotype. Interactions between genes and environments (GxE) increase the heritability estimate when genes condition the effects of environments on the phenotype by
influencing study members' vulnerability or resistance to risky environments. When estimates of heritability are very large, this encourages researchers to look for GxE and GrE effects on that phenotype. Following from our taxonomic theory of life-course-persistent antisocial behavior, the genetic component of variation in antisocial behavior measured in early childhood ought to comprise not only the direct effects of genes, but also the effects of correlations between vulnerability genes and risky environments, and interactions between them as well.

Studies have documented the existence of correlations between children's genetic predisposition toward antisocial behavior and their rearing environments, or GrE. Specifically, three adoption studies and one twin study have now shown that children with a genetic liability for antisocial behavior tend to evoke harsher discipline from their parents than do children lacking this liability (Ge et al., 1996; Jaffe, Caspi, Moffitt, Polo-Tomas, et al., 2004; O'Connor et al., 1998; Riggins-Caspers et al., 2003). However, to our knowledge, no research has examined whether this evocative child effect in turn exacerbates levels of the children's antisocial behavior when they are followed over time. Evidence of exacerbation is necessary to answer whether gene-environment correlations increase estimates of heritability for antisocial behavior over and above any direct effects of genes.

There is somewhat better evidence that interactions between genetic and environmental risk, or GxE, contribute to the heritability of life-course-persistent antisocial behavior. We tested GxE in two studies of one of the strongest risk factors for persistent antisocial behavior: child maltreatment. The first study used data from our Environmental-risk Longitudinal Twin Study (called E-risk) to test whether the effect of maltreatment on risk for antisocial behavior was strongest among 5-year-olds who were at high genetic risk for it. The E-risk Study follows a representative 1994 to 1995 cohort of 1,116 British twin pairs and their families (Moffitt & the E-Risk Study Team, 2002). Each child's genetic risk for conduct problems was estimated as a function of his or her cotwin's Conduct Disorder status and the pair's zygosity. For example, a child whose monozygotic (MZ) twin already had Conduct Disorder was deemed at highest genetic risk, whereas a child whose dizygotic (DZ) twin had Conduct Disorder was at lower genetic risk, and a child whose MZ twin was free from conduct problems was at the lowest genetic risk of all. Results showed that the effects of maltreatment were most detrimental for the children at high genetic risk. The experience of maltreatment was associated with an increase of 2% in the probability of a Conduct Disorder diagnosis among young children at low genetic risk for Conduct Disorder but an increase of 24% among children at high genetic risk (Jaffe et al., 2005).

Our second study used data from the male members of the Dunedin cohort to test whether this general GxE effect could be specified for a particular candidate gene. We used Dunedin men's DNA to genotype a polymorphism in the gene encoding the neurotransmitter-metabolizing enzyme monoamine oxidase A (MAOA). We selected this gene because it was known to be functional in the brain and it had earlier been related to aggression in mice and in a human family pedigree. MAOA genotype moderated the effect of maltreatment (Caspi et al., 2002). Of the severely maltreated children with a genotype conferring low levels of MAOA expression, 85% developed one or more antisocial outcomes, as measured at different stages in the life course (e.g., a childhood diagnosis of Conduct Disorder up to age 15, conviction for violent crimes between ages 17 and 26, antisocial personality traits at age 26, and diagnosed Antisocial Personality Disorder at age 26). In contrast, despite being maltreated, children with a genotype conferring high levels of MAOA expression were less likely to develop antisocial problems. Although individuals having the combination of low-activity MAOA genotype and maltreatment were only 12% of the birth cohort, they accounted for 44% of the cohort's convictions for violent crimes. This finding has been replicated and extended in the Virginia Study of Adolescent Twin Development. This study was able to control for the parents' antisocial histories to rule out the possibility that a family liability toward aggression jointly influenced parents' maltreating behavior and children's antisocial outcome (Foley et al., 2004). Together, the E-risk, Dunedin, and Virginia studies of GxE illustrate how the high heritability of the life-course-persistent form of antisocial behavior is played out in the transactions between a genetically vulnerable child and high-risk environment that were specified in the theory of life-course-persistent antisocial development.

What Research Is Needed?

It would be useful to ascertain the genetic and environmental architecture of individual differences in trajectories of antisocial behavior over time. Such trajectories could be derived in longitudinal studies of twins by applying semiparametric mixture modeling tools to repeated measures of antisocial behavior (for explanations of these models, see Nagin et al., 1995; Nagin & Tremblay, 2001a; Roeder et al., 1999). The theory would predict stronger monozygotic
twin similarity for membership in a childhood-onset, life-course-persistent trajectory, but less twin similarity for membership in an adolescent-onset trajectory. Because no twin studies to our knowledge have yet followed a sample of twins from childhood to adulthood while measuring antisocial behavior, a study of twin similarity in developmental trajectories of antisocial behavior is yet in the future. However, genetically informative studies could examine as proxies the elements of crime careers that characterize the life-course-persistent type: early onset of antisocial acts, recidivistic physical aggression, and psychopathic personality traits. One study has attempted this approach and reported that genetic influence is very strong among children who exhibit a callous, unemotional personality style by age 7 years (Viding et al., 2005).

IS A THIRD GROUP NEEDED? CHILDHOOD-LIMITED AGGRESSIVE CHILDREN MAY BECOME LOW-LEVEL CHRONIC CRIMINAL OFFENDERS WITH PERSONALITY DISORDERS

The original theoretical taxonomy asserted that two prototypes, life-course-persistent and adolescence-limited offenders, account for the preponderance of the population’s antisocial behavior and thus warrant the lion’s share of attention by theory and research. However, our analyses revealed a small group of Dunedin study males who had exhibited extreme, pervasive, and persistent antisocial behavior problems during childhood, but who surprisingly engaged in only low to moderate delinquency during adolescence from age 15 to 18, not extreme enough to meet criteria for membership in the life-course-persistent group (Moffitt et al., 1996). Like the life-course-persistent offenders, they had extremely undercontrolled temperaments as 3-year-olds (Moffitt et al., 1996), and in childhood they, too, suffered family adversity and parental psychopathology and had low intelligence (unpublished analyses). The existence of a small group of boys who exhibit serious aggression in childhood but are not notably delinquent in adolescence has been replicated in the Pittsburgh Youth Survey, where they were called “childhood-limited” antisocial children (Raine et al., 2005). In the Pittsburgh cohort, too, these boys had many risk factors, including family adversity, parental psychopathology, and severe neuropsychological deficits. This group was a surprise to the theory, because the theory argued that an early-onset chain of cumulative interactions between aggressive children and high-risk environments will perpetuate disordered behavior. On that basis, we had predicted that “false positive subjects, who meet criteria for a stable and pervasive antisocial childhood history and yet recover (eschew delinquency) after puberty, should be extremely rare” (Moffitt, 1993, p. 694). When we discovered this group, we optimistically labeled it the “recovery group” (Moffitt et al., 1996). Many researchers, we among them, hoped that this group would allow us to identify protective factors that can be harnessed to prevent childhood aggression from persisting and becoming more severe. However, our study of this group has revealed no protective factors.

Researchers testing for the presence of the life-course-persistent and adolescence-limited types have since uncovered a third type that replicates across longitudinal studies, first identified in trajectory analyses of a British cohort (Nagin et al., 1995). This third group of offenders have been labeled “low-level chronics” because they have been found to offend persistently but at a low rate from childhood to adolescence (Fergusson et al., 2000) or from adolescence to adulthood (D’Unger, Land, McCall, & Nagin, 1998; Nagin et al., 1995). Persuaded by these findings, we followed up the so-called recovery group in the Dunedin cohort at age 26 to see if they might fit the low-level chronic pattern as adults. We found that recovery was clearly a misnomer, as their modal offending pattern over time fit a pattern referred to by criminologists as “intermittency,” in which some offenders are not convicted for a period but then reappear in the courts (Laub & Sampson, 2001). This Dunedin group’s long-term offending pattern closely resembles that of the low-level chronic offender.

Anticipating true recoveries from serious childhood Conduct Disorder to be extremely rare, the taxonomic theory had argued that teens who engage in less delinquency than predicted on the basis of their childhood conduct problems might have off-putting personal characteristics that excluded them from the social peer groups in which most delinquency happens. Consistent with this prediction, a group in the Oregon Youth Study, who showed high levels of antisocial behavior at age 12 that decreased thereafter, scored low as adolescents on a measure of involvement with pro-delinquency peers (Wiesner & Capaldi, 2003). In the Dunedin cohort followed up to age 26, the members of this low-level chronic group, unlike other cohort men, were often social isolates; their informants reported that they had difficulty making friends, none had married, few held jobs, and many had diagnoses of Agoraphobia and/or Social Phobia. Almost all social phobics meet criteria for
Avoidant, Dependent, and/or Schizotypal Personality Disorder (Alnaes & Torgeresen, 1988), and we speculate that men in this group may suffer from these isolating personality disorders. As many as one-third of this group had diagnosable depression, their personality profile showed elevated neuroticism, and their informants rated them as the most depressed, anxious men in the cohort. This pattern, in which formerly antisocial boys develop into depressed, anxious, socially isolated men, resembles closely a finding from a British longitudinal study of males followed from ages 8 to 32. In that study, too, at-risk antisocial boys who became adult “false positives” (committing fewer crimes than predicted) had few or no friends, held low-paid jobs, lived in dirty home conditions, and had been described in case records as withdrawn, highly strung, obsessive, nervous, or timid (Farrington, Gallagher, Morley, St. Ledger, & West, 1988).

Robins (1966) is often quoted as having said that half of conduct problem boys do not grow up to have antisocial personalities. Such quotations are intended to imply that early conduct problems are fully malleable and need not be a cause for pessimism. However, less often quoted is Robins’ observation that conduct problem boys who do not develop antisocial personalities generally suffer other forms of maladjustment as adults. This is an assertion of “multifinality” in the poor outcomes of at-risk children (Cicchetti & Cohen, 1995). In the Dunedin birth cohort, 87 boys had childhood conduct problems (i.e., 47 in the life-course-persistent and 40 in the so-called recovery group). Of these 87 males, only 15% (n = 13) seemed to have truly recovered as adults, escaping all adjustment problems measured in the study at age 26. Taken together, findings from Dunedin and the studies by Farrington and Robins are consistent with our taxonomic theory’s original assertion that childhood-onset antisocial behavior is virtually always a prognosticator of poor adult adjustment.

What Research Is Needed?

Several studies have detected an unexpected group, variously labeled “recoveries,” “childhood-limited,” or “low-level chronic offenders,” depending on how long the cohort was followed. However, few studies have been able to shed any light on their personal characteristics. The characteristics revealed so far are suggestive of Avoidant, Dependent, Schizotypal Personality Disorders and/or low intelligence, but these outcomes have not been directly measured in adulthood. It is important to know if this group has adult psychopathology to test the theory’s assertion that serious childhood-onset antisocial behavior reliably predicts long-term maladjustment.

IS A FOURTH GROUP NEEDED? ADULT-ONSET ANTISOCIAL BEHAVIOR

On the basis of examining official data sources, some investigators have suggested that significant numbers of offenders first begin to offend as adults (Eggleston & Laub, 2002; Farrington, Ohlin, & Wilson, 1986). This would appear to challenge our developmental taxonomy’s assertion that two groups, life-course-persistent and adolescence-limited, suffice to account for the majority of antisocial participation across the life course. However, the observation that many antisocial individuals are adult-onset offenders appears to be largely an artifact of official measurement. Estimates of the age at which antisocial behavior begins depend on the source of the data. For example, in the Dunedin study, only 4% of boys had been convicted in court by age 15 years, but 15% had been arrested by police by age 15, and 80% had self-reported the onset of illegal behaviors by age 15 (Moffitt et al., 2001, chap. 7). This suggests that official data lag behind the true age of onset by a few years. Similar findings have emerged from other studies in other countries. For example, a Canadian survey showed that self-reported onset antedated conviction by about 3.5 years (Loebber & LeBlanc, 1990), and a U.S. survey showed that self-reported onset of “serious” delinquency antedated the first court contact by 2.5 years and onset of “moderate” delinquency antedated the first court contact by 5 years (U.S. Office of Juvenile Justice and Delinquency Prevention, 1998). In the Seattle Social Development cohort, the self-reported onset of crime antedated the first court referral by 2.4 years, and the study estimated that the average offender committed 26 crimes before his official crime record began (Farrington et al., 2003). These comparisons of data sources suggest that investigations relying on official data will ascertain age of onset approximately 3 to 5 years after it has happened. A 3- to 5-year lag is relevant because most studies have defined adult-onset offenders as those whose official crime record began at or after age 18 years (Eggleston & Laub, 2002).

It also is useful to note that whereas the 18th birthday may have marked adulthood for young people born before 1960, the 18th birthday falls only midway between puberty and adulthood for contemporary generations. This shift emerged because contemporary generations are experiencing a more protracted adolescence, lasting until the mid-
20s (Arnett, 2000) or even into the early 30s for the cohort born after 1970 (Ferril, Byrner, & Wadsworth, 2003; Furstenberg, Cook, Sampson, & Slap, 2002). Although adult-onset crime begins at age 18 in legal terms, in developmental terms for contemporary cohort samples, it begins sometime after age 25.

In contrast to studies using official crime records, self-report cohort studies show that fewer than 4% of males commit their first criminal offense after age 17 (Elliott, Huizinga, & Menard, 1989). Self-report studies of American and European cohorts agree (Junger-Tas, Terlouw, & Klein, 1994). By age 18, virtually all of the Dunedin study members had already engaged in some form of illegal behavior at some time, according to their self-reports (Moffitt et al., 2001). Only 9% of Dunedin males and 14% of females remained naïve to all delinquency by age 18, and only 3% of males and 5% of females first offended as an adult, between ages 18 and 21. These findings carry an important lesson for methodology in developmental research into antisocial behavior. "Adult-onset" offenders cannot be defined for study with any certainty unless self-reported data are available to rule out juvenile onset prior to participants' first official contact with the judicial system. When self-report data are consulted, they reveal that onset of antisocial behavior after adolescence is extremely rare. This conclusion extends to serious and violent offending (Elliott, 1994).

One way to ascertain whether adult-onset offenders constitute a significant group for study is to apply semiparametric modeling techniques (Nagin, 1999; Nagin & Tremblay, 2001a; Roeder et al., 1999) to identify trajectories within a population-representative cohort of individuals whose behavior has been followed into adulthood. Three studies have done so. The Dunedin study identified no adult-onset trajectory in self-reports of delinquency from ages 7 to 26 years (see Figure 15.1). The Oregon Youth Study identified no adult-onset trajectory in self-reports of offending from ages 12 to 24 years (Wiesner & Capaldi, 2003). The Cambridge Longitudinal Study identified no adult-onset trajectory in official crime records followed to age 32 for a cohort born in the 1950s (Nagin et al., 1995).

The original theoretical taxonomy asserted that two prototypes, life-course-persistent and adolescence-limited offenders, can account for the preponderance of the population's antisocial behavior. After more than 10 years of research, this assertion appears to be correct. Some studies of the taxonomy have reported an adult-onset group (e.g., Kratzer & Hodgins, 1999). However, these studies used official crime data, and thus most of their adult-onset offend-

![Figure 15.1 Twenty-year trajectories of conduct disorder symptoms among 525 Blunden males.](image)

ers would probably be revealed as adolescent-onset if self-report data were available. These so-called adult-onset offenders can probably be accommodated by the adolescence-limited theory because when studied, the alleged adult-onset group has not differed from ordinary adolescent offenders (Eggleston & Laub, 2002). Moreover, like adolescence-limited offenders, adult-onset offenders' crime careers tend to be brief and not serious (Farrington et al., 1986). In our view, the existence of individuals whose official crime record begins after age 18 does not constitute a threat to the taxonomy.

**THE HYPOTHESIS THAT ADOLESCENCE-LIMITED ANTISOCIAL BEHAVIOR IS INFLUENCED BY THE MATURITY GAP AND BY SOCIAL MIMICRY OF ANTISOCIAL MODELS**

The original theory asserted that individual differences should play little or no role in the prediction of short-term adolescent offending careers. Instead, the strongest predictors of adolescence-limited offending should be peer delinquency, attitudes toward adolescence and adulthood reflecting the maturity gap [such as a desire for autonomy], cultural and historical contexts influencing adolescence, and age. (Moffitt, 1993, p. 695)

Most research on the taxonomy to date has focused on testing hypotheses about the etiology of life-course-persistent offenders. Unfortunately, adolescence-limited offenders have been relegated to the status of a contrast group,
and the original hypotheses about the distinct etiology of adolescent-onset offending have not captured the research imagination. This is unfortunate because adolescent-onset offenders are quite common (one quarter of both males and females as defined in the Dunedin cohort) and their antisocial activities are not benign. They are found among adjudicated delinquents as well as in the general population (Scholte, 1999). Moreover, even if adolescence-limited individuals commit fewer violent offenses than life-course-persistent individuals, the size of the adolescence-limited group is much larger than the size of the life-course-persistent group, and as a result the adolescence-limited group can be expected to account for an important share of a society’s serious and violent offenses. In Dunedin, life-course-persistent men (10% of the cohort) accounted for 53% of the cohort’s 554 self-reported violent offenses at age 26, but adolescence-limited men (26% of the cohort) accounted for 29% of the cohort’s violent offenses, a nontrivial amount of violence (Moffitt, Caspi, Harrington, & Milne, 2002).

Do adolescents find the maturity gap psychologically aversive, and does this motivate their newfound interest in delinquency? Aguilar et al. (2000) discovered that adolescent-onset delinquents experienced elevated internalizing symptoms and perceptions of stress at age 16, which may be consistent with the taxonomy’s assertion that these adolescents experience psychological discomfort during the maturity gap. The theory suggested that this discomfort motivated adolescents to engage in antisocial behavior to seem older. In a study of the Gluecks’ sample, adolescents’ concerns about appearing immature increased their likelihood of delinquency (Zebrowitz, Andreoletti, Collins, Lee, & Blumenthal, 1998). One interesting ethnographic study has made use of the maturity gap to explain korteliralli, the street-racing alcohol youth culture of Finland (Vaaranen, 2001). The Victoria Adolescence Project studied 452 adolescents and their parents to examine how young people negotiate the maturity gap (Galambos, Barker, & Tilton-Weaver, 2003). This study identified a group of 25% of adolescents who exhibited a cluster of characteristics they called “pseudo-maturity.” These adolescents, relative to their age cohort, were characterized by more advanced biological pubertal status, older subjective age (“I feel a lot older than my age”), elevated perceptions of self-reliance, more wishes to emulate older brothers (but not sisters), more older friends, a greater desire to be older (“I would like to look a lot older than my age”), more involvement in pop culture, and less involvement in school but more involvement with peers. This cluster was not associated with SES level. The study concluded that for a large proportion of teens, pubertal maturation brings about a poor fit between their developmental stage and their social environment: “They are caught in the maturity gap” (Galambos et al., 2003, p. 262). Parent and self-reports confirmed that this pseudo-mature group of teenagers engaged in elevated rates of problem behaviors, as expected by the theory of adolescence-limited delinquency.

Do adolescence-limited teenagers want to be more like life-course-persistent offenders? The theory of adolescence-limited delinquency borrowed the concept of “social mimicry” from the field of ethology to explain how adolescents might mimic the antisocial behavior of life-course-persistent antisocial boys in their midst in an effort to attain the mature status embodied in the antisocial lifestyle. New developmental research has shown that when ordinary young people age into adolescence, they begin to admire good students less and to admire aggressive, antisocial peers more (Bukowski, Sippola, & Newcomb, 2000; Luthar & McMahon, 1996; Rodkin, Farmer, Pearl, & Van Acker, 2000). One sociometric study that followed 905 children from age 10 to 14 reported that the association between physical aggression and being disliked by peers dissolved during this age period; as they grew older, the teenagers came to perceive their aggressive age-mates as having higher social status and more influence (Cillessen & Mayeux, 2004). Moreover, during adolescence, young people who place a high value on conforming to adults’ rules become unpopular with their peers (Allen, Weissberg, & Hawkins, 1989).

Our Dunedin studies documented that an increase in young teens’ awareness of peers’ delinquency antedates and predicts onset of their own later delinquency (Casp, Lynam, Moffitt, & Silva, 1993). We also showed that the adolescence-limited path is more strongly associated with delinquent peers, as compared to the life-course-persistent path (Jeglum-Bartusch et al., 1997; Moffitt & Caspi, 2001). However, one study that traced peer affiliation trajectories concluded that peers were as influential for childhood-onset persistent offenders as for adolescent-onset offenders (LaCourse, Nagin, Tremblay, Vitaro, & Claes, 2003). In contrast, others have shown that delinquent peer influences directly promote increases in delinquency, specifically among young males whose antisocial behavior begins in adolescence (Simons, Wu, Conger, & Lorenz, 1994; Vitaro, Tremblay, Kerr, Pagani, & Bukowski, 1997). In contrast, these same studies suggest that among males whose antisocial behavior begins in childhood, the direction of influence runs the other way; the child’s own early antisocial behavior promotes increases at adolescence in the number of delinquent peers who selectively affiliate.
with him. This is consistent with our life-course-persistent theory’s assertion that during adolescence, life-course-persistent antisocial boys become “magnets” for peers who wish to learn delinquency.

The most direct test of the adolescence-limited etiological hypothesis was carried out in the Youth in Transition Survey of 2,000 males (Piquero & Brezina, 2001). This study was introduced to the literature with lyrics from a song entitled “Eighteen” by rocker Alice Cooper that express the ennui of the maturity gap: “I’m in the middle without any plans, I’m a boy and I’m a man.” The study tested the hypothesis that desires for autonomy promoted adolescent-onset offending. It found that, as predicted, the offenses committed by adolescence-limited delinquents were primarily rebellious (not physically aggressive) and that this rebellious offending was accounted for by the interaction between maturational timing and aspects of peer activities that were related to personal autonomy. However, one measure of youth autonomy in this study did not predict offending.

It is important to acknowledge that alternative accounts of late-onset delinquency have been put forward. In particular, Patterson and Yoerger (1997) outlined a learning model in which decreases in parents’ monitoring and supervision when their children enter adolescence cause adolescents to begin offending. We had argued that although parents’ monitoring and supervision were certainly negatively correlated with adolescent-onset delinquency, the direction of cause and effect was unclear, and our adolescence-limited theory would say that this correlation arises because teens’ desires to gain autonomy via delinquency motivate them to evade their parents’ supervision (Moffitt, 1993, p. 693). A longitudinal study of 1,000 Swedish 14-year-olds and their parents suggested that our interpretation is correct (Kerr & Stattin, 2000). Adolescents actively controlled their parents’ access to information about their activities, and teens who took part in deviant behavior limited their parents’ capacity to monitor them. The study showed that parents’ efforts to supervise and monitor were not very effective in controlling their teenagers’ activities, and could even backfire if teens felt controlled.

**What Research Is Needed?**

Clearly, there is not very much research testing whether measures of the maturity gap and social mimicry can account for adolescence-limited delinquency, so any new studies with this aim would add to our understanding. Agnew (2003) offers a cogent breakdown of maturity gap elements that can be tested. Short-term longitudinal studies of young teens might ask if a developmental increase in attitudes rejecting childhood and favoring autonomy is correlated with a growing interest in and approval of illicit activities. Moreover, there is the curious fact that life-course-persistent antisocial individuals are rejected by peers in childhood but later become more popular with peers in adolescence. The theory of social mimicry predicted this shift in popularity, but more longitudinal research following individuals’ changes in social standing is needed to understand it fully. Finally, we should consult historical and anthropological work to ascertain if historical periods and cultures characterized by a clearly demarcated transition from childhood dependency to adulthood rights and responsibilities are also characterized by relatively low levels of delinquency and adolescent rebelliousness.

**THE HYPOTHESIS THAT ABSTAINERS FROM DELINQUENCY ARE RARE INDIVIDUALS WHO ARE EXCLUDED FROM NORMATIVE PEER GROUP ACTIVITIES IN ADOLESCENCE**

If, as the theory says, adolescence-limited delinquency is normative adaptational social behavior, then the existence of teens who abstain from delinquency requires an explanation. In other words, if ordinary teens take up delinquent behavior, then teens who eschew delinquency must be extraordinary in some way. The original theory speculated that teens committing no antisocial behavior would be rare and that they must have either structural barriers that prevent them from learning about delinquency, no maturity gap because of early access to adult roles, or personal characteristics unappealing to other teens that cause them to be excluded from teen social group activities (Moffitt, 1993, pp. 689, 695). As noted earlier, research has shown that during adolescence, young people who place a high value on conforming to adults’ rules become unpopular with their peers (Allen et al., 1989).

We have studied male abstainers in the Dunedin cohort. Consistent with the rarity prediction, the Dunedin cohort contained only a very small group of males who avoided virtually any antisocial behavior during childhood and adolescence; abstainers were fewer than 10% of the cohort (Moffitt et al., 1996). The very small size of this group has been confirmed in other samples. Only 13% of 17-year-olds in the National Longitudinal Survey of Youth replied that they had never done any of the survey’s 13 offense items (Piquero, Brezina, & Turner, in press). Two longitudinal cohort studies used a theory-free method to characterize heterogeneous trajectories within repeated measures
of aggressive behavior: Nagin and Tremblay (1999) detected an abスター trajectory from childhood to adolescence containing very few males, and Wiesner and Capaldi (2003) detected an abスター trajectory from adolescence to adulthood containing even fewer males (5%).

The small group of Dunedin abstainers described themselves at age 18 on personality measures as extremely over-controlled, fearful, interpersonally timid, and socially inept, and they were latecomers to sexual relationships (i.e., virgins at age 18). Dunedin abstainers fit the profile that Shedler and Block (1990) reported for youth who abstained from drug experimentation in a historical period when it was normative: overcontrolled, not curious, not active, not open to experience, socially isolated, and lacking social skills. Dunedin abstainers were unusually good students, fitting the profile of the compliant good student who, during adolescence, can become unpopular with peers (Allen et al., 1989; Bukowski et al., 2000). Other studies have suggested that abstention from delinquency and substance use during adolescence is associated with feeling socially isolated from peers (Dunford & Elliott, 1984), having few friends (Farrington & West, 1993), or being a loner (Tolone & Tieman, 1990). Such findings prompted Shedler and Block (1990, p. 627) to comment that abstention is “less the result of moral fiber or successful prevention programs than the result of relative alienation from peers and a characterological overcontrol of needs and impulses.”

Dunedin’s age-26 follow-up data confirmed that the teenage abstainers did not become so-called adult-onset offenders (Moffitt, Caspi, et al., 2002). Although their teenage years had been socially awkward for them, their style became more successful in adulthood. As adults they retained their self-constrained personality, had virtually no crime or mental disorder, were likely to have settled into marriage, were delaying children (a desirable strategy for a generation needing prolonged education to succeed), were likely to be college-educated, held high-status jobs, and expressed optimism about their own future.

Another study of abstainers from delinquency was conducted using 1,600 17-year-olds from the 1997 National Longitudinal Survey of Youth (Piquero, Brezina, et al., in press). Consistent with theoretical prediction, relative to participants in delinquency the abstainers were few in number, more closely monitored by their parents, more attached to teachers, and less physically mature, reported less autonomy, dated less, and were less involved with friends who drank, smoked, tried drugs, and cut classes. However, an unexpected new finding was that abstainers were not wholly friendless; rather, they reported that they had prosocial peers who “go to church regularly,” “plan to go to college,” and “participate in volunteer work.” This study also attempted to test the theory’s prediction that abstainers have personalities that make them unattractive to peers, using an item called “sadness/depression” intended to assess a morose, uncheerful style unlikely to appeal to peers. However, the study found that sadness/depression was correlated with delinquent participation, not abstention. This test was ambiguous because the depression item probably did not measure the overcontrolled, incurious, timid, socially inept personality style thought to preclude delinquency. Thus, this study provided some modest support for the taxonomy’s view of abstainers as a minority that exists outside the social scene that creates opportunities for delinquency among the teen majority. Moreover, the study suggested the provocative new finding that abstainers do have friends, who are prosocial like themselves.

What Research Is Needed?

To our knowledge, our finding that abstainers are social introverts as teens remains to be confirmed or discounted by another study directly designed to test this hypothesis. Adolescent sociometric studies might ask if delinquent abstention is indeed correlated with unpopularity and social isolation. Further study of abstainers is critical for testing the hypothesis that adolescence-limited offenders’ delinquency is normative adaptational behavior by ordinary young people.

THE HYPOTHESIS THAT LIFE-COURSE-PERSISTENT AND ADOLESCENCE-LIMITED DELINQUENTS DEVELOP DIFFERENT PERSONALITY STRUCTURES

The original theory hypothesized the following about the development of life-course-persistent offenders:

Over the years, an antisocial personality is slowly and insidiously constructed, and accumulating consequences of the youngster’s personality problems prune away options for change. . . . A person-environment interaction process is needed to account for emerging antisocial behavior, but after some age, will the “person” main effect alone predict adult outcome? (Moffitt, 1993, p. 684)

Our Dunedin studies of adolescents’ personality characteristics measured at age 18 showed that the life-course-persistent path was differentially associated with weak bonds to family and with the psychopathic personality
traits of alienation, callousness, and impulsivity. In contrast, the adolescence-limited path at age 18 was differentially associated with a tendency to endorse unconventional values and with a personality trait called "social potency" (Moffitt et al., 1996). We assessed personality traits 10 years later at age 26, this time using not only self-reports but reports from informants who knew the Dunedin study members well (Moffitt, Caspi, et al., 2002). The self- and informant-reports concurred that the life-course-persistent men had more negative emotionality (were more stress-reactive, alienated, and aggressive) and they were less agreeable (had less social closeness, were more callous) compared to adolescence-limited men. Life-course-persistent men were no longer particularly impulsive at age 26, but the adolescence-limited men were still somewhat elevated on this scale at age 26. It appears from these repeated Dunedin assessments that the life-course-persistent pathway leads to a disordered antisocial personality structure resembling the psychopath: aggressive, alienated, and callous. Adolescence-limited men, in contrast, are unconventional, valuing spontaneity and excitement.

In another study, 4,000 California Youth Authority (CYA) inmates were given the California Personality Inventory (Gough, 1987) in the 1960s and then followed up into the 1980s (Donnellan, Ge, & Wenk, 2002). Taxonomy comparison groups were defined as early starters versus later starters, and as chronic adult arrestees versus those arrested less often. The early-starter, chronic arrestees could be discriminated by extreme personality scale scores, in particular low communality, little concern with impression, irresponsibility, low control of emotions, low achievement motivation, low socialization, low tolerance (hostile, distrustful), and low well-being. Early starters also scored higher than late starters on the Schizophrenia and Hypomania scales of the Minnesota Multiphasic Personality Inventory (Dahlstrom, Welsh, & Dahlstrom, 1972), two scales that measure a tendency to think in a confused and suspicious way (Ge, Donnellan, & Wenk, 2003). Using different instruments and different informants, these CYS findings echo our Dunedin findings, in which life-course-persistent offenders were disagreeable and high on negative emotionality.

What Research Is Needed?

To our knowledge, the personality correlates of the taxonomy have been examined in only two samples (Dunedin and CYA), so the finding of differential personality structures remains to be verified by wider replication. Moreover, the unanticipated Dunedin finding that many adolescence-limited offenders are unconventional excitement-seekers raises the question of whether this approach-oriented personality style is present prospectively before they take up delinquency and is an individual-difference risk factor for adolescent onset. If so, that was not anticipated by the theory. Childhood temperament studies that have measured the approach style might follow up their participants to ask if approach predicts adolescent-onset delinquency. Longitudinal research is also needed to determine if and when the antisocial personality style becomes set, that is, able to predict adult antisocial outcomes alone, without any further environmental input.

THE HYPOTHESIS THAT LIFE-COURSE-PERSISTENT DEVELOPMENT IS DIFFERENTIALY ASSOCIATED IN ADULTHOOD WITH SERIOUS OFFENDING AND VIOLENCE

The original theory predicted that life-course-persistent offenders, as compared to adolescence-limited offenders, would engage in a wider variety of offense types, including "more of the victim-oriented offenses, such as violence and fraud" (Moffitt, 1993, p. 695).

By the time the Dunedin cohort reached age 18, we reported that the life-course-persistent pathway was differentially associated with conviction for violent crimes (Jeglum-Bartusch et al., 1997; Moffitt et al., 1996), and the adolescence-limited pathway was differentially associated with nonviolent delinquent offenses (Jeglum-Bartusch et al., 1997). These Dunedin findings are buttressed by reports from other samples that physical aggression usually begins in childhood and seldom begins in adolescence (e.g., Brame, Nagin, & Tremblay, 2001). Moreover, we had shown that preadolescent antisocial behavior that was accompanied by neuropsychological deficits predicted greater persistence of crime and more violence up to age 18 (Moffitt et al., 1994).

Our follow-up at age 26 confirmed that life-course-persistent men as a group particularly differed from adolescence-limited men in the realm of violence, including violence against the women and children in their homes. This finding was corroborated with large effect sizes by data from multiple independent sources, including self-reports, informant reports, and official court conviction records (Moffitt, Caspi, et al., 2002). In a comparison of specific offenses, life-course-persistent men tended to specialize in serious offenses (carrying a hidden weapon, assault, robbery, violating court orders), whereas
adolescence-limited men specialized in nonserious offenses (theft less than $5, public drunkenness, giving false information on application forms, pirating computer software). Life-course-persistent men accounted for 5 times their share of the cohort's violent convictions. Thus, although they were a small group (10% of males), they accounted for 43% of the cohort's officially sanctioned violent crime.

Domestic violence against women and children at home was specifically predicted to be an outcome of the life-course-persistent group (Moffitt, 1993). At the age-26 Dunedin follow-up, this group's scores were elevated on self-reported and official conviction measures of abuse toward women, both physical abuse (e.g., beating her up, throwing her bodily) and controlling abuse (e.g., stalking her, restricting her access to her friends and family). Because the Dunedin cohort has been interviewed repeatedly about illicit behaviors for many years, study members now trust the study's guarantee of confidentiality and can be asked questions about hitting children, with the expectation of valid responses. Life-course-persistent men were the most likely to report that they had hit a child out of anger, not in the course of normal discipline. Our finding that life-course-persistent offenders perpetrated more domestic violence was supported by the Christchurch study's finding that young adults with childhood-onset antisocial behavior engaged in significantly more violence against partners than did those with adolescent-onset antisocial behavior (Woodward, Fergusson, & Horwood, 2002). Similarly, a study of New York parolees reported that those defined as life-course-persistent based on a childhood-onset offense record engaged in twice as much domestic violence as parolees with an adolescent-onset offense record (Mazerolle & Maahs, 2002).

In general, a large empirical literature shows that the strongest long-term predictors of violence are the same predictors implicated by our theory of life-course-persistent offending: early-onset antisocial behavior, neurodevelopmental risk factors, and family risk factors (for a review, see Farrington, 1998). Moreover, research comparing violent crime versus general nonviolent delinquency has shown that violence is differentially predicted by birth complications (Raine et al., 1997), minor physical anomalies (Arseneault et al., 2000), difficult temperament (Henry, Caspi, Moffitt, & Silva, 1996), and cognitive deficits (Piquero, 2001), each of which is a hypothetical risk for life-course-persistent development (for a review, see Raine, 2002). The Christchurch study reported that people with serious childhood-onset conduct problems, compared to children without conduct problems, engaged in 10 times more violent crime by age 25 (Fergusson, Horwood, & Ridder, 2005). The Patterns of Care Study of 1,715 service users ages 6 to 17 years compared childhood-onset versus adolescent-onset Conduct Disorder cases and reported that the childhood-onset group committed significantly more bullying but not more of the other physically aggressive Conduct Disorder symptoms (McCabe et al., 2001). However, this study did not have an adult follow-up. Lahey and colleagues (1998) reported more physical aggression associated with adolescent-onset than with childhood-onset Conduct Disorder.

What Research Is Needed?

The literature makes it clear that neurodevelopmental and family risks predict violence when it is measured on a continuum, but only a few studies have compared the adult violent outcomes of groups defined on the basis of early versus late antisocial onset. In addition, research is needed to clarify why life-course-persistent offenders are more violent. Our theory implies that verbal cognitive deficits may limit their options for handling conflict (a neuropsychological explanation), that they may have learned in their family that violence is an effective way to manage conflict (a social-cognition explanation), and that broken attachment bonds lead to alienation from their potential victims (an attachment explanation; Moffitt, 1994; Moffitt & Caspi, 1995). All of these explanations specify early childhood as a critical period influencing adult violence. But which, if any, of these explanatory processes are correct? Research using designs that control for genetic transmission of a predisposition to aggression in families has now documented that experiences in the family do promote childhood-onset aggression through processes that are environmentally mediated. Environmental effects on children's aggression have now been documented for exposure to parents' domestic violence (Jaffee, Moffitt, Caspi, Taylor, & Arseneault, 2002), being reared by an antisocial father (Jaffee, Moffitt, Caspi, & Taylor, 2003), being reared by a depressed mother (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005), being a recipient of maternal hostility (Caspi et al., 2004), and being a victim of child maltreatment (Jaffee, Caspi, Moffitt, & Taylor, 2004). These studies controlled for familial liability to psychopathology, suggesting that the risk factors influence children through environmental experience. This information gives fresh impetus for research to uncover how these experiences are mediated via the child's thoughts and emotions to produce persistent aggression. Research is needed on mediating developmental processes, because findings will point to targets for intervention.
THE HYPOTHESIS THAT CHILDHOOD-ONSET ANTISOCIAL BEHAVIOR WILL PERSIST INTO MIDDLE ADULTHOOD, WHEREAS ADOLESCENT-ONSET ANTISOCIAL BEHAVIOR WILL DESIST IN YOUNG ADULTHOOD

Inherent in the name "life-course-persistent" is the assertion that the antisocial activities of these individuals will persist across the life course. Though the rest of the population may decrease its antisocial participation as it ages, the life-course-persistent individuals should remain at the top of the heap on antisocial behaviors. Thus, the taxonomy accepts that antisocial participation declines markedly in midlife; nonetheless, it expects rank-order stability, particularly on age-relevant measures of antisocial activity. To test the differential desistance prediction, it is necessary to follow a cohort’s antisocial behavior from childhood to adulthood, but only a few studies have done this.

We followed up the Dunedin cohort at age 26 (Moffitt, Caspi, et al., 2002) to test hypotheses critical to this part of the theory: Childhood-onset antisocial behavior, but not adolescent-onset antisocial behavior, should be associated in adulthood with antisocial personality and continued serious antisocial behavior that expands into maladjustment in work life and victimization of partners and children (Moffitt, 1993, p. 695). Followed to age 26, the adolescent-onset delinquents at 26 were still engaging in elevated levels of property offending and they had financial problems, but they did not show a pattern of serious offending. Interestingly, the adolescent-onset delinquents self-reported problems with mental health and substance dependence, but these difficulties were not corroborated by informants who knew them well. Consistent with the taxonomy’s predictions, the childhood-onset delinquents at age 26 were the most elevated on psychopathic personality traits, mental health problems, substance dependence, numbers of children sired, financial problems, work problems, domestic abuse of women and children, and drug-related and violent crimes.

In a study of 4,000 CYA inmates followed into their 30s, significantly more early starters than later starters continued offending past age 21, past age 25, and past age 31. Moreover, early onset and low cognitive ability significantly predicted which inmates continued to offend past age 31 (Ge, Donnellan, & Wenk, 2001). A different study of CYA offenders looked in depth at predictors of criminal career duration among 377 paroles released on average at age 24 and followed for 12 years (Piquero, Brame, & Lynam, 2004). This study found that criminal career duration was predicted by low tested cognitive abilities and by the interaction between childhood poverty status and cognitive ability. Similarly, a large Swedish study reported less crime in adulthood among offenders who possessed positive personal characteristics resembling the characteristics of Dunedin adolescence-limited offenders (Stattn, Romelsjo, & Stenbacka, 1997).

These findings were obtained using groups of adolescence-limited and life-course-persistent males defined by applying commonsense clinical cutoffs (e.g., Moffitt et al., 1996). However, in the past decade, new analytic methods have become available for ascertaining whether distinctive trajectories exist within a population of individuals whose behavior has been measured repeatedly during development (Nagin, 1999; Nagin & Tremblay, 2001a; Roeder et al., 1996). These new semiparametric methods offer several advantages over the clinical cutoffs approach. First, the methods are agnostic with respect to taxonomic theories, and thus results are relatively free from investigator bias. Second, the methods can search a longitudinal data set to ask whether there is indeed more than one developmental trajectory in it, as a taxonomy implies. Third, they can ascertain the relative goodness of fit of competing models having 1, 2, 3, 4, or more trajectories to ascertain whether the taxonomic theory has specified the right number of developmental subtypes in the population. Fourth, they generate output from the best-fitting model that reveals whether its trajectories rise and fall at ages specified by the theory. Fifth, they generate output about which study participants belong to which trajectory, making it possible to ascertain whether each trajectory group approximates its population prevalence as specified by the theory. It is important to keep in mind that what researchers put into the method determines what they can get out, and therefore testing the taxonomy of life-course-persistent and adolescence-limited antisocial behavior calls for representative samples, repeated measures taken at informative ages from childhood to adulthood, and measures of antisocial behavior that capture its heterotypic continuity across developmental periods. In these respects, the Dunedin data set, although not perfect, was pretty good fodder for the semiparametric method.

We applied this method to counts of Conduct Disorder symptoms assessed (via self-, mother-, and teacher-reports) for 525 male study members at ages 7, 9, 11, 13, 15, 18, 21, and 26 years. Conduct Disorder symptoms are fighting, bullying, lying, stealing, cruelty to people or animals, vandalism, and disobeying rules; three such symptoms earn a formal diagnosis. The model that best fit the Dunedin data detected the following groups (see Figure 15.1). A
life-course-persistent group, 7% of the cohort, had a fairly stable high trajectory, exhibiting between 4 and 7 antisocial symptoms at every age from 7 to 26 years. This group had more symptoms than any of the other groups at every age. A group whose trajectory resembled an adolescence-limited pattern began with 2 symptoms at age 7 but increased to a peak of 4.5 symptoms at age 18, and then decreased on a slight downward trajectory to 3.5 symptoms at age 26. A recovery group, 21% of the cohort (similar to the childhood-limited or low-level chronic groups described in an earlier section of this chapter), began with 6 symptoms at age 7 but decreased steadily with age and had only 1 symptom by ages 21 and 26. An abstainer group, 11%, had less than 1 symptom on average at every age. Two further trajectory groups were identified. The first of these took an adolescence-limited shape, but at a low level, and the second took a recovery shape, but also at a low level. For illustrative purposes, in Figure 15.1 these two groups were collapsed into a consistently low group, 47% of the cohort, which had 1 to 2 symptoms on average at each age. Thus, the best-fitting model bore a not unreasonable resemblance to the taxonomy. Differential outcomes for the trajectory groups mirrored the outcomes for the clinically defined Dunedin groups (Moffitt, Caspi, et al., 2002). Males on the adolescence-limited trajectory were still engaging in property offending and substance abuse but not serious offending at age 26. Males on the life-course-persistent trajectory were the most elevated at age 26 on mental health problems and substance dependence, numbers of children sired, financial and work problems, domestic abuse of women and children, and drug-related and violent crimes.

Other cohort studies have applied trajectory analysis to repeated measures of antisocial behavior from childhood to adulthood. A British longitudinal study followed official crime records for a 1950s birth cohort of 400 men to age 32 and detected chronic and adolescence-limited trajectories that showed the expected differential desistance (Nagin et al., 1995). Unexpectedly, offenders defined as adolescence-limited had desisted from criminal offending according to their official police records, but according to their self-reports they continued into their 30s to drink heavily and get into fights. The South Holland epidemiological study followed 2,000 Dutch children from age 4 to 30 years (Bongers, Koot, van der Ende, Donker, & Verhulst, in press). This study reported two trajectories of young people with high levels of externalizing problems, as assessed by the CBCL (Achenbach, 1985). One trajectory was normative and distinguished by increasing truancy and alcohol and drug use, but did not markedly increase the risk of adult offending. The other trajectory was characterized by increasing oppositional behavior and hot temper and was associated with elevated risk of serious and violent adult offending. Low trajectories were also detected.

The Rutgers Health and Human Development Project followed its longitudinal sample into adulthood and reported a test of the taxonomy using nonparametric mixture modeling to detect trajectory groups (H. R. White, Bates, & Buyske, 2001). However, White et al.’s figure 1, showing delinquency trajectories for the resulting groups, suggests that the group labeled “persistent” in this study was in reality adolescence-limited, because this group’s trajectory showed very low levels of offending at ages 12 and 28 but a very pronounced adolescent offending peak at age 18. This sample may not have contained life-course-persistent members, because it was recruited via random telephone dialing with an initial 17% rate of refusal to the phone call and afterward a 52% completion rate for enrollment in data collection. Families with life-course-persistent risk characteristics are known to be difficult to engage as research participants (Farrington, Gallagher, Morley, St. Ledger, & West, 1990), and therefore they were probably among those who did not take part in the Rutgers study. Given the strong possibility that groups were mislabeled in this study, it is unclear what to make of it vis-à-vis the taxonomy.

The Oregon Youth Study applied trajectory analysis to 200 males followed from age 12 to 24 (Wiesner & Capaldi, 2003). In addition to the abstainer trajectory and the decreasing trajectory discussed in earlier sections of this chapter, the analysis also yielded a group whose antisocial behavior was chronically at the cohort’s highest level (life-course-persistent?) and a group whose antisocial behavior increased somewhat from age 12 to a peak at 19, and then decreased from age 20 to 24 (adolescence-limited?). It is not clear that Wiesner and Capaldi would agree with our characterization of their groups; indeed, they used different labels for them. In any case, although these two groups seemed fairly similar in late adolescence, they diverged at the study’s age 23 to 26 outcome point, with the chronic group showing much higher levels of alcohol use, drug use, and depression symptoms, as well as more adult antisocial behavior (Wiesner, Kim, & Capaldi, in press).

One clear shortcoming of the available longitudinal data that have been used to test for the presence of life-course-persistent versus adolescence-limited subtypes is that the data are “right-hand censored”; in other words, study participants have generally been followed only until their 20s or 30s. What is needed is a cohort that represents the general population and that has been followed through the age period of risk for most criminal offending, up to midlife.
Such a cohort does not yet exist. However, in the absence of the ideal representative cohort, there is one important study that warrants our focus. Sampson and Laub (2003) reported a follow-up of half of the Gluecks' sample, those who were adolescent inmates in Massachusetts in the 1940s. The authors constructed a unique database of official criminal records for almost 500 men, covering the period from age 7 years to the end of each offender’s life, up to age 70 years. The study was noteworthy for collecting nationwide FBI records and for attending to artifacts in crime records arising from periods of incarceration or the offender’s premature death. The authors' analyses were motivated by their skepticism about the idea of prospectively predicting a group of offenders who will account for a disproportionate amount of society's serious crime. Sampson and Laub reported two findings from the study that they believed challenged this idea. First, they found that almost all of the men in the Gluecks' sample desisted from criminal offending sooner or later. Second, they found heterogeneity in adulthood crime career patterns within the sample of adolescent inmates, and they found that this heterogeneity was not explained by measures of childhood risk. Because the Sampson and Laub publication was represented as a challenge to the life-course-persistent taxonomy, we must take a closer look at whether or not these two findings discredit the taxonomy. In so doing, it is useful to consider the nature of the sample studied by Sampson and Laub. According to the taxonomy, virtually all of the men studied would have been regarded as candidates for the life-course-persistent subtype. They had been incarcerated as young adolescents in reform schools, a status reserved at that time for a very small fraction of a state's youth, those having established already by adolescence the most serious, persistent records of deviance that could not be controlled by parents or schools. It is well documented that as a group, the boys had backgrounds of marked family adversity, social disadvantage, and childhood antisocial conduct. Sampson and Laub note details about the sample that fit the life-course-persistent pattern, such as low mean IQ and mean first arrest at 11.9 years. Thus, this sample born in 1924 to 1932 probably comprised, relative to the much larger population of Boston males their age, a small subgroup who had started on the life-course-persistent pathway.

Sampson and Laub's (2003, p. 577) first finding was that the men in the Gluecks' sample desisted from criminal offending sooner or later: "Aging out of crime appears to reflect a general process." Unfortunately, Sampson and Laub misrepresented the taxonomy's prediction; they set up a straw prediction to test that life-course-persistent offenders should carry on committing crimes at the same high rate from adolescence through old age and to their death. Clearly, this was never implied by the taxonomy, because it acknowledged the populationwide process of aging out of crime and explained that the term life-course-persistent antisocial behavior did not require crime in old age, but referred to the persistence of antisocial personality characteristics or antisocial behaviors within the family (Moffitt, 1993, p. 680). The taxonomy's actual prediction was that delinquents like the Gluecks' would continue offending well beyond the age when most young men in their cohort population desisted. The study followed only reform school boys, and thus it could not provide comparative data on crime careers for Boston men born 1924 to 1932, but it is known that desistance from delinquency in young adulthood was the norm for this cohort, which came of age in the postwar era of near full employment. In contrast to that norm, 84% of the study men were arrested between ages 17 and 24, 44% were arrested in their 40s, 23% were arrested in their 50s, and 12% were arrested in their 60s. The reform school sample's mean crime career length was 25.6 years. It seems reasonable to believe that such remarkable statistics do not also describe the rest of the male population of Boston. Thus, the study's results seem reasonably consistent with the taxonomy's prediction that boys who begin life on the life-course-persistent pathway will have unusually extended offending careers, thereby accounting for more than their share of the crime rate.

Sampson and Laub's (2003) second finding was of heterogeneity in adulthood crime career patterns within the Gluecks' sample. Again, the alternative hypothesis seems to be a straw man. The alternative would be that males who spent their youth and early adulthood on the life-course-persistent pathway can show no variation in subsequent offending during midlife and aging over a span of many years. Such uniformity is implausible, and the taxonomic theory did not make such a prediction. Within the Gluecks' sample, six trajectories emerged from a semiparametric group-based modeling analysis. Thus, the men, all of whom began on the life-course-persistent pathway, varied subsequently in their age at desistance from crime and in their rate of offending up to the point of their desistance. Of particular importance, child and family characteristics did not discriminate among these six trajectories. On the one hand, this failure of discrimination is not surprising given that the cohort members' childhood backgrounds were almost uniformly high-risk. On the other hand, this finding suggests that to the extent that different crime careers emerge during midlife within a group of life-course-persistent men, concurrent life experiences must account for the divergence. This would constitute an interesting extension to
the taxonomic theory on a topic it did not originally address: heterogeneity within life-course-persistent delinquents in the ways they age out of crime.

This study by Sampson and Laub (2003) was well executed and well intentioned. The authors were concerned about practitioners who have reified the life-course-persistent idea, treating it as if it describes a group having hard boundaries, made up of individual children who are easy to identify in early childhood and who deserve radical interventions to avert their inevitable destiny as predatory criminals. The authors’ concern is well placed, and their efforts to dissuade such reification are laudable. To their credit, the authors point out that “the current bandwagon . . . is not consistent with the logic of Moffitt’s actual argument” (p. 576). Nonetheless, to make their points, the authors inadvertently had to misrepresent the original taxonomy as having made predictions that it did not make. Here we set the record straight. Life-course-persistent delinquents do not have to be arrested for illegal crimes steadily up to age 70, but they do have to maintain a constellation of antisocial attitudes, values, and proclivities that affect their behavior toward others. Life-course-persistent delinquents do not have to live exactly the same crime trajectory as they age out of crime; it is interesting to learn how their lives diverge. Laub and Sampson (2003) are leading the way in researching these questions using qualitative as well as quantitative methods.

What Research Is Needed?

Overall, our theory’s prediction that childhood-onset antisocial behavior persists longer into adulthood than adolescent-onset delinquency seems to be on fairly solid empirical footing. It has been known for decades that early onset of offending predicts a longer duration of crime career, and this association was recently affirmed by two careful reviews (Gendreau, Little, & Goggin, 1996; Krohn, Thornberry, Rivera, & LeBlanc, 2001). Nonetheless, the adolescence-limited groups in the Dunedin cohort and other cohorts continued to experience some adjustment problems as adults, and we need research to understand what accounts for this. The original taxonomy put forward the hypothesis that we should expect some adolescence-limited delinquents to recover to good adult adjustment later than others, and that this age variation might be explained by snares such as a conviction record that harms job prospects (Moffitt, 1993, p. 691). The idea is that engaging in even limited delinquency as a young person can diminish the probability of subsequent good outcomes, particularly if one is caught and sanctioned. Also important is the information emerging from the work of Laub and Sampson (2003) pointing to marked heterogeneity within the life-course-persistent group in middle and late life, suggesting that research into midlife turning-point experiences is needed. Overall, longitudinal studies are needed that follow the life-course-persistent, low-level chronic, abstainer, and adolescence-limited groups to reveal the very long-term implications of their experiences in the first 2 decades of life.

GENDER: THE HYPOTHESIS THAT MOST FEMALE ANTISOCIAL BEHAVIOR IS THE ADOLESCENCE-LIMITED TYPE

The original statement of the taxonomy asserted that the theory describes the behavior of females as well as it describes the behavior of males. The full text of the theory that 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 verbal and motor milestones, hyperactivity, learning disabilities, reading failure, and childhood conduct problems. . . . Most girls lack the personal diathesis elements of the evocative, reactive, and proactive person/environment interactions 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. (pp. 39–40)

The original theory thus proposed that (1) fewer females than males would become delinquent (and conduct disordered) overall and that (2) among delinquents, the percentage who are life-course-persistent would be larger for males than females. Following from this, (3) the majority of delinquent females will be of the adolescence-limited type, and (4) their delinquency will have the same causes as adolescence-limited males’ delinquency.

These predictions were borne out in the Dunedin cohort (Moffitt & Caspi, 2001; Moffitt et al., 2001). As predicted, the male:female difference was very large for the life-course-persistent form of antisocial behavior (10:1) but negligible for the adolescence-limited form (1.5:1). Childhood-onset females had high-risk neurodevelopmental and family backgrounds, but adolescent-onset females did not, which documented that females and males on the same trajectories share the same risk factors. We have described the elements of the adolescence-limited causal pathway among Dunedin females, showing that each girl’s delinquency onset is linked to the timing of her own puberty (Moffitt et al., 2001), that delinquent peers are a necessary condition for onset of delinquency among adolescent girls (Caspi et al., 1993; Caspi & Moffitt, 1991), and that an intimate relationship with an offender promotes girls’ antisocial behaviors (Moffitt et al., 2001).

Few empirical tests of this taxonomy have compared how females and males fit the two developmental trajectories, but it appears that our findings about Dunedin females are broadly consistent with these previous studies. Ferguson and colleagues (2000, 2002), studying the Christchurch sample (n = 1,000), 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, versus only 2:1 for late-onset subjects. Kratzer and Hodgins (1999), studying a Swedish cohort (n = 13,000), found similar childhood risk factors for males and females in the life-course-persistent group, and the male to female ratio was 15:1 for early-onset, versus only 4:1 for late-onset subjects. Mazeroille et al. (2000), studying a Philadelphia cohort (n = 3,655), reported that early onset signaled persistent and diverse offending for males and females alike. A longitudinal study of 820 girls analyzed with a semiparametric mixture model found a stable, highly antisocial group, but this group contained only 1.4% of the girls (Cote, Zoccolillo, Tremblay, Nagin, & Vitaro, 2001).

All studies concur that females are seldom childhood-onset- or life-course-persistent-type (the exception is Aguilar et al., 2000, whose early-onset group had as many girls as boys).

The application of our developmental taxonomy to females was questioned by Silverthorn and Frick (1999), who put forward a contrasting, female-specific theory. They suggested that despite the fact that girls’ onset of antisocial behavior is delayed until adolescence, this adolescent-onset pathway in girls is not analogous to the adolescence-limited pathway in boys. Instead, this theory argued, all delinquent girls have the same high-risk causal backgrounds as life-course-persistent males. To evaluate this alternative hypothesis, we conducted analyses in the Dunedin study (Moffitt et al., 1996) using self-reports, mothers’ reports, and teachers’ reports of antisocial behavior to define girls on the adolescence-limited and life-course-persistent paths. This study resulted in two findings: First, the vast majority of female delinquents fit the adolescence-limited, late-starter pattern; second, the childhood backgrounds of these females who exhibited adolescent-onset antisocial behavior were like the backgrounds of adolescence-limited delinquent boys, normative and not pathological. The proposal from Silverthorn and Frick has also been tested in the African American Philadelphia cohort of the National Collaborative Perinatal Project (N. A. White & Piquero, in press), using official crime records to define adolescence-limited and life-course-persistent offenders. This study, too, reported that most female offenders followed the adolescence-limited pattern, and that female and male adolescence-limited offenders scored similarly, and better than life-course-persistent offenders, on measures of family and individual risk such as childhood cognitive ability. Similar conclusions were reached in the Patterns of Care Study of 300 children receiving services for Conduct Disorder (McCabe, Rodgers, Yeh, & Hough, 2004). Because the African American cohort and the Patterns of Care sample were selected for high-risk status, childhood-onset girls were found at higher prevalence than in the Dunedin cohort, which represents the general population.

These accumulating findings suggest that the two theories of the origins of life-course-persistent and adolescence-limited offending describe the developmental course of antisocial behavior across the sexes and irrespective of sex. Because few females have the risk factors for life-course-persistent development, this theory explains the wide sex difference in serious, persistent antisocial behavior. Because the risk factors for adolescence-limited offending are equal-opportunity, this theory explains the sex
similarity for nonserious, transient delinquency during the teenage years (Moffitt et al., 2001).

What Research Is Needed?

The dearth of gender comparisons originates from a pragmatic circumstance. An ideal test of this theory requires a large representative (nonclinical, nonadjudicated) sample followed longitudinally from childhood to adulthood with repeated measures of antisocial behavior. To date, few such studies have included females. It would be useful to have a large sample including both life-course-persistent and adolescence-limited girls in sufficient numbers to test all components of our theory. The new longitudinal study of Pittsburgh girls may help in this regard (Hipwell et al., 2002).

RACE: THE HYPOTHESIS THAT BOTH LIFE-COURSE-PERSISTENT AND ADOLESCENCE-LIMITED DEVELOPMENTAL PROCESSES ARE EXACERBATED BY SOCIETAL RACE PREJUDICE

The original statement of the taxonomy asserted that the theory applies to ethnic minority populations as well as to Whites. The discussion of race was published in a book that is not easy to obtain (Moffitt, 1994). Therefore, we paraphrase the original statement:

In the United States, the crime rate for black Americans is higher than the crime rate for whites. The race difference may be accounted for by a relatively higher prevalence of both life-course persistent and adolescence-limited subtypes among contemporary African Americans. Life-course persistent antisocials might be anticipated at elevated rates among black Americans because the putative root causes of this type are elevated by institutionalised prejudice and by poverty. Among poor black families, prenatal care is less available, infant nutrition is poorer, and the incidence of exposure to toxic and infectious agents is greater, placing infants at risk for the nervous system problems that research has shown to interfere with prosocial child development. To the extent that family bonds have been loosened and poor black parents are under stress ... and to the extent that poor black children attend disadvantaged schools ... for poor black children the snowball of cumulative continuity may begin rolling earlier, and it may roll faster downhill. In addition, adolescence-limited crime is probably elevated among black youths as compared to white youths in contemporary America. If racially segregated communities provide greater exposure to life-course persistent role models, then circumstances are ripe for black teens with no prior behavior problems to mimic delinquent ways in a search for status and respect. Moreover, black young people spend more years in the maturity gap, on average, than whites because ascendency to valued adult roles and privileges comes later, if at all. Legitimate desirable jobs are close to many young black men; they do not often shift from having "little to lose" to having a "stake in conformity" overnight by leaving schooling and entering a good job. Indeed, the biological maturity gap is perhaps best seen as an instigator of adolescent-onset delinquency for black youths, with an economic maturity gap maintaining offending into adulthood. (p. 39)

Thus, the taxonomy expected that both life-course-persistent and adolescence-limited causal processes should work the same way in African American and White American groups, but any excess of offending among poor African American youth could be attributed to an excess of the risk factors for both delinquent subtypes. Our research with the Pittsburgh Youth Survey has documented that childhood risk factors associated with life-course-persistent offending (low IQ and impulsive undercontrol) are related to early-onset, frequent delinquent offending and physical aggression among Black and White males alike (Caspi et al., 1994; Lynam et al., 2000; Lynam, Moffitt, & Stouthamer-Loeb, 1993). However, these studies have not specifically divided Pittsburgh delinquents into childhood-versus adolescent-onset comparison groups (because the cohort of the Pittsburgh study having IQ and personality data was not followed beyond age 13).

We know of only a few studies that have directly compared life-course-persistent versus adolescence-limited offender patterns across races, and they seem to offer opposing findings. Donnellan et al. (2000) designated the taxonomy groups in 2,000 young adult CYA inmates. On a comprehensive set of measures of cognitive ability, life-course-persistent offenders scored below adolescence-limited offenders. However, this predicted finding of differential cognitive risk applied to adjudicated Whites and Hispanics but not to adjudicated African Americans. The opposite pattern of race differences emerged from a different study of 377 CYA offenders released on average at age 24 and followed for 12 years (Piquero et al., 2004). This study found that African American parolees had worse childhood poverty and lower cognitive test scores than White parolees, the interaction between poverty and low cognitive ability predicted longer criminal career duration, and this prediction was stronger among African American than White parolees.

A study of the Baltimore sample of the National Collaborative Perinatal Project was able to test for race differ-
ences in the etiological process hypothesized to underlie life-course-persistent antisocial behavior (Piquero, Moffitt, & Lawton, in press). Results showed that several variables helped to explain differences between Whites and Blacks in the level of chronic offending. However, although Black participants had higher mean levels of risk factors than Whites, the developmental processes predicting chronic offending were the same across groups defined by race. Specifically, low birthweight in combination with adverse familial environments predicted chronic offending from adolescence to age 33 among Whites and African Americans alike, although the effect size reached statistical significance among only African Americans, a pattern opposite that reported by Donnellan et al. (2000). Most, but not all, of these studies of African American cohorts fit our theory’s notion that causal developmental processes are the same across racial groups, but that African Americans end up with higher levels of crime because they begin the processes with higher levels of risk factors.

One further set of studies is relevant. A study of the Philadelphia sample of the National Collaborative Perinatal Project, which includes only African American families, showed that childhood measures of school discipline problems and low tested IQ predicted a more serious offending career (Piquero & Chung, 2001). In this same Philadelphia African American cohort, low scores on an achievement test of cognitive ability predicted early onset and subsequent persistence of offending (McGloin & Pratt, 2003), and neuropsychological test scores at ages 7 to 8 years and 13 to 14 years predicted patterns of life-course-persistent offending to age 39 (Piquero & White, 2003). In the Providence sample of the National Collaborative Perinatal Project, Piquero and Buka (2002) found the expected higher prevalence of offenders among African Americans compared to Whites. However, crime career patterns such as the concentration of crimes in a few offenders and the significant prediction of adult crime from chronic juvenile delinquency, applied equally well to both races. It has been shown that earlier onset of delinquent offending among African American males predicts a more serious crime career (Piquero & Chung, 2001) and a longer duration of crime career (Piquero et al., 2004), which suggests that the life-course-persistent pattern applies to Black samples as well as White samples.

What Research Is Needed?
The theory asserted that life-course-persistent and adolescence-limited processes should apply to both Whites and ethnic minorities (neurodevelopmental and family risks should predict the earlier onset, greater seriousness, and longer duration of antisocial behavior across racial groups, and the maturity gap and peer influences should predict late onset across racial groups). Together, these causal processes should be able to account for the elevated prevalence of delinquency among ethnic minorities because minority group members experience higher levels of both sets of risk factors. Rowe, Vazsonyi, and Flannery (1994) provide an explanation of how to test this.

Most of the studies of African Americans cited here used cohorts defined in the period between 1955 and 1980. Of course, only cohorts defined some time ago can yield information about the duration and seriousness of crime careers followed into the 3rd decade of life, but research on more contemporary cohorts is also needed.

There is one caveat: If the maturity gap lasts longer for African American young men than for Whites, this would make it difficult to distinguish the life-course-persistent from adolescence-limited groups in Black samples on the basis of chronic offending into adulthood. Therefore, researchers examining contemporary cohorts of ethnic minorities should operationalize the life-course-persistent and adolescence-limited groups using other distinguishing features, such as early childhood onset of conduct problems, antisocial personality traits, or recidivistic youth violence.

RESEARCH NEEDED ON OTHER HYPOTHESES
Before 1993, virtually no research compared delinquent subtypes defined on a developmental basis, but now this research strategy has become almost commonplace. Many research teams have assessed representative samples with prospective measures of antisocial behavior from childhood to adulthood, and this has enabled comparisons based on age of onset and persistence. Now that the requisite databases are available, other hypotheses derived from the original taxonomic theory need to be tested.

First, we suggested that childhood measures of antisocial behavior in longitudinal studies should be more highly correlated with adult measures than with adolescent measures of antisocial behavior (Moffitt, 1993, p. 695). This hypothesis derived from the taxonomy’s assertion that the people participating in antisocial behavior in childhood and adulthood are the same people, but they are joined in adolescence by other people whose reasons for participation are different. If childhood measures did correlate with adult measures better than with adolescent measures, it would be a surprising violation of the so-called longitudinal law that measures taken closer together in time are
more strongly correlated than measures taken further apart in time. In the Dunedin study, we asked how closely age-3 undercontrolled temperament was associated with later antisocial diagnostic outcomes (controlling for sex). Age-3 undercontrol was a significant predictor of adult diagnoses 2 decades later: OR = 3.1 (CI = 1.3–7.5) for age-26 Antisocial Personality Disorder and OR = 4.1 (CI: 2.0 to 8.4) for age-21 Conduct Disorder. However, the associations between age-3 temperament and adolescent diagnoses were not significant: OR = 1.7 (CI = 0.9 to 3.4) for age 15 Conduct Disorder and OR = 1.8 (CI = 0.9 to 3.8) for age 18 Conduct Disorder. The South Holland epidemiological study reported a similar finding: Antisocial behaviors assessed at ages 6 to 11 years predicted antisocial behaviors at ages 12 to 17 years only weakly (OR = 2.5), but predicted antisocial behavior at ages 20 to 25 years strongly (OR = 6.7; Donker, Smeenk, van der Laan, & Verhulst, 2003). More such tests of the hypothesis that childhood antisocial behavior predicts adult outcome better than adolescent outcome are needed.

Second, we speculated that adolescence-limited offenders must rely on peer support for crime, but life-course-persistent offenders should be willing to offend alone (although in adolescence, they serve as magnets for less expert offenders; Moffitt, 1993, p. 688). To our knowledge, this hypothesis has not been systematically examined.

Third, we suggested that snares (such as a criminal record, incarceration, addiction, and truncated education without credentials) should explain variation in the age at desistance from crime during the adult age period, particularly among adolescence-limited offenders (Moffitt, 1993, p. 691). One study reported that alcohol and cannabis dependence can ensnare young people in an antisocial lifestyle; most individuals’ personal curve of antisocial participation from age 18 to 26 generally grows downward, but a spell of substance dependence temporarily deflects this curve upward (Hussong, Curran, Moffitt, Caspi, & Carrig, 2004). More such tests of the hypothesis that snares mediate continuing offending are needed.

Fourth, we asserted that the two groups would react differently to turning-point opportunities: Life-course-persistent offenders would selectively get undesirable partners and jobs and would in turn expand their repertoire into domestic abuse and workplace crime, whereas adolescence-limited offenders would get good partners and jobs and would in turn desist from crime (Moffitt, 1993, p. 695). One study showed that the potential turning point of school grade retention could prompt antisocial behavior among ordinary boys, but in contrast, retention had little effect on boys who had been on a chronic life-course-persistent trajectory since childhood; the authors speculated that the chronic boys’ antisocial behavior was already so ingrained that it was difficult to influence for good or for bad (Nagin, Pagani, Tremblay, & Vitaro, 2003). More such tests of the hypothesis that antisocial history moderates turning points are needed.

Fifth, we put forward a new prediction here, that the life-course-persistent antisocial individual will be at high risk in midlife for poor physical health, cardiovascular disease, and early disease morbidity and mortality (Moffitt, 2001). We based this prediction on our observation that life-course-persistent antisocial behavior is associated in adolescence with factors that are known to predict disease morbidity in midlife (Gallo & Matthews, 1999; McEwen, 2002; Repetti, Taylor, & Seeman, 2002). Such risk factors include health risk behaviors (e.g., heavy smoking, unprotected sex), high levels of stress (e.g., conflictual relationships, family dissolution, financial insecurity), and personality traits associated with physiological vulnerability (e.g., hostility, stress-reactivity, alienation). Our preliminary analyses of health indicators collected when the Dunedin study members were 26 years old has revealed that, compared to the adolescence-limited men, the life-course-persistent antisocial men already had somewhat lower high-density lipoprotein and significantly poorer tested cardiorespiratory aerobic fitness (Moffitt et al., 2005). Follow-up assessments of longitudinal studies of antisocial development should measure more physical health outcomes.

CONCLUSIONS

After 10 years of research, what can be stated with some certainty is that the hypothesized life-course-persistent antisocial individual exists, at least during the first 3 decades of life. Consensus about this group has emerged from all studies that have applied trajectory-detection analyses to a representative cohort sample having longitudinal repeated measures of antisocial behavior. Tremblay et al. (2004) detected a “high physical aggression” group constituting 14% of Canadian children followed from age 17 to 42 months. Broidy et al. (2003) detected a “chronic aggressive” group constituting 3% to 11% of children followed from age 6 to 13 years in six different cohorts from three countries; Maughan et al. (2001) detected a “stable high aggressive” group constituting 12% of North Carolina youth followed from ages 9 to 16 years. Brame et al. (2001) detected a “high chronic aggressive” group constituting 3% of Canadian youth followed from ages 6 to 17 years.
Raine et al. (2005) detected a “life-course persistent path” group that constituted 13% of Pittsburgh youth followed from ages 7 to 17 years. Fergusson et al. (2000) detected a “chronic offender” group constituting 6% of Christchurch youth followed from ages 12 to 18 years. Chung et al. (2002) detected a “chronic offender” group constituting 7% of Seattle youth followed from ages 13 to 21 years. Wiesner and Capaldi (2003) detected a “chronic high-level” group constituting 16% of Oregon youth followed from ages 12 to 24 years. Moffitt et al. (this chapter) detected a “high-persistent” group that constituted 7% of Dunedin young people followed from ages 7 to 26 years. Nagin et al. (1995) detected a “high-level chronic” group that constituted 12% of London males followed from ages 10 to 32 years. So far as we know, no research team that has looked for a persistent antisocial group has failed to find it.

A number of other postulates from the taxonomy have received modest or mixed empirical support, but overall they appear to be supported:

- Life-course-persistent antisocial behavior emerges from early neurodevelopmental and family adversity risk factors, but adolescence-limited delinquency does not.
- Genetic etiological processes contribute more to life-course-persistent than adolescence-limited antisocial development.
- Childhood-limited aggressive children, if followed to adulthood, become low-level chronic criminal offenders with personality disorders.
- Abstainers from delinquency are rare individuals who become unpopular with teen peers.
- Life-course-persistent and adolescence-limited delinquents develop different personality structures by adulthood.
- Life-course-persistent development is differentially associated with serious offending and violence in adulthood.
- Life-course-persistent antisocial development is almost exclusively male, whereas most female antisocial behavior is of the adolescence-limited type.

Some findings have received beginning support, but more research is needed:

- Adolescence-limited antisocial behavior is influenced by the maturity gap between childhood and adulthood and by social mimicry of antisocial role models.
- Childhood-onset antisocial behavior persists at least into middle adulthood, whereas adolescent-onset antisocial behavior desists in young adulthood.

Some predictions from the taxonomy have not been tested sufficiently:

- Life-course-persistent antisocial individuals will be at high risk in midlife for poor physical health, cardiovascular disease, and early disease morbidity and mortality.
- Adolescence-limited offenders must rely on peer support for crime, but life-course-persistent offenders should be willing to offend alone (although in adolescence, they serve as magnets for less expert offenders).
- Snares (such as a criminal record, incarceration, addiction, and truncated education without credentials) should explain variation in the age at desistance from crime during the adult age period, particularly among adolescence-limited offenders.
- The two groups should react differently to turning-point opportunities: Adolescence-limited offenders should get good partners and jobs that help them to desist from crime, whereas life-course-persistent offenders should selectively get undesirable partners and jobs and in turn expand their repertoire into domestic abuse and workplace crime.

It is pleasing that the taxonomy has generated interest and research. Some findings have been faithful to the hypotheses originally formulated. Other findings have pointed to important revisions needed to improve the fit between the taxonomy and nature, and some findings raise serious challenges to aspects of the taxonomy. All three kinds of findings are much appreciated. Obviously, there is still a lot of work to do.

REFERENCES


Life-Course-Persistent versus Adolescence-Limited Antisocial Behavior


