

Evidence for a Genetic Etiology of Early-Onset Delinquency

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Age at onset of antisocial behavior discriminates persistent and transitory offenders. The authors proposed that early-onset delinquency has an underlying genetic influence that manifests in problems related to inhibition, whereas late-onset delinquency is more environmentally mediated. To test these notions, they selected 36 early starters, 86 late starters, and 25 nondelinquent controls from a large sample of 11-year-old twins and compared them on several measures related to inhibition and a peer group measure. As expected, early starters had more psychological, behavioral, and emotional problems related to inhibition than late starters and controls. A longitudinal analysis indicated an increase in antisocial behavior among peers of late starters shortly before their delinquency onset. Family history data and a twin analysis provided evidence of greater genetic influence on early-onset than late-onset delinquency.

Age at onset is the single best predictor of severity and course of antisocial behavior (see Farrington et al., 1990, for a review). Delinquency that begins in adolescence is thought to be transitory (Farrington et al., 1990; Moffitt, 1993). What causes some children to be aggressive, unruly, or antisocial from an early age and persist in this behavior, whereas others wait until after puberty sets in to break rules and get into trouble? Answers to these kinds of questions may lead to better interventions for delinquency and perhaps to more effective strategies for dealing with long-term criminality.

In their review of the behavioral genetic literature on delinquency, DiLalla and Gottesman (1989) proposed two subgroups: continuous antisocials and transitory delinquents. DiLalla and Gottesman suggested that the "continuous" subtype had a stronger genetic influence (accounting for the continuity of antisocial behavior), whereas the "transitory" subtype was largely a product of the environment. In 1993, Moffitt laid out a theoretical explanation for (essentially) the two delinquency subtypes proposed by DiLalla and Gottesman. Moffitt called her offender subtypes *adolescence-limited* and *life-course-persistent*. These labels reflect the essence of their difference: the persistence of antisocial behavior across the life span. Moffitt estimated that as much as 80% of adolescents exhibit some form of antisocial behavior, whereas persistent antisocial behavior occurs among only 5% of the population. Moffitt suggested that adolescence-limited offending is normative and results from an affiliation with and mimicking of antisocial peers; life-course-persistent offending is more akin to a disorder and may

be linked to a host of neuropsychological and behavioral deficits. (We used the term *early starters* to refer to continuous antisocials or life-course-persistent offenders and *late starters* to refer to transitory delinquents or adolescence-limited offenders.)

Antisocial behavior in children is associated with a number of biological and psychological characteristics (some of which may also differentiate early and late starters). As compared to non-antisocial children, antisocial children tend to show poor verbal ability (Lahey et al., 1995; Moffitt, 1990; Moffitt & Silva, 1988; White et al., 1994), impulsivity (White et al., 1994), high neuroticism (negative emotionality) and low constraint (Tremblay, Pihl, Vitaro, & Dobkin, 1994), and higher rates of attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD; Biederman, Munir, & Knee, 1987; Faraone et al., 1995; Lahey et al., 1995; Loeber, Green, Keenan, & Lahey, 1995; Moffitt, 1990; White, Moffitt, Earls, Robins, & Silva, 1990). Antisocial boys also exhibit autonomic hypoactivity (Borkovec, 1970; Fox & Lippert, 1963; Lippert & Senter, 1966; Raine, Venables, & Williams, 1990; Siddie, Nicol, & Foggitt, 1973), which may indicate low sympathetic nervous system arousal and perhaps relate to higher levels of sensation seeking and behavioral disinhibition. In addition to these characteristics, several studies support a connection between delinquency and antisocial behavior in peers (e.g., Jessor, Van Den Bos, Vanderryn, Costa, & Turbin, 1995; Rowe & Flannery, 1994; Simons, Wu, Conger, & Lorenz, 1994). To date, these characteristics have not been examined together on a sample of early and late starters, and thus it is not clear whether these factors differentiate or perhaps unite the subtypes.

Patterson (1986) proposed one of the earliest psychological models of why some boys show early-onset, persistent antisocial behavior, and his theory may account for some of the characteristics found among early starters. Patterson posited that early starters cultivate inaction from their parents through defiant, coercive behavior that is followed by school failure and affiliation with antisocial peers (after rejection by prosocial peers). Thus, from Patterson's theory one might predict higher rates of ODD, lower verbal IQ scores, and a more antisocial peer group for early starters as compared to late starters and nondelinquents. However,

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many other characteristics of early starters are not as readily predicted from Patterson's theory.

If not solely related to a dysfunctional environment, then why do early starters tend to exhibit characteristics such as impulsivity, inattention and overactivity, oppositionality and disobedience, low behavioral control, negative emotionality, and perhaps decreased autonomic arousal? We propose that early starters have an underlying genetic component to the etiology of their antisocial behavior that manifests as behavioral and personality problems related to inhibition. Researchers have reported that early-onset delinquents are highly disinhibited. We propose that the particular behavioral and personality problems related to inhibition observed among early starters are each influenced by genetic factors and thus may be part of an underlying genetic liability toward early-onset, persistent antisocial behavior. Evidence suggests a substantial genetic influence on impulsivity (Nadder, Silberg, Eaves, Maes, & Meyer, 1998; Plomin, 1976; Sherman, Iacono, & McGue, 1997; Stevenson & Fielding, 1985; but also see Plomin, Coon, Carey, & DeFries, 1992); negative emotionality and constraint (e.g., Billig, Hershberger, Iacono, & McGue, 1996; Finkel & McGue, 1997; Tellegen et al., 1988); symptoms of ADHD and ODD (Eaves et al., 1997; Edelbrock, Rende, Plomin, & Thompson, 1995; Faraone et al., 1995; Nadder et al., 1998; Sherman et al., 1997; Silberg et al., 1996); and autonomic activity measures such as changes in sweat gland activity referred to as skin conductance (SC; Kotchoubeï, 1987; Lykken, Iacono, Haroian, McGue, & Bouchard, 1988; Rust, 1984).

Reviews of behavioral genetic studies of delinquency (e.g., DiLalla & Gottesman, 1989) suggest a substantial role for environmental influences. As Moffitt (1993) suggested, antisocial peers probably exert significant environmental influence on late-onset delinquents, but antisocial peers are also part of the early starters' environment (Patterson, 1986; Simons et al., 1994). In addition to deviant peers, early antisocial behavior may be fostered in family environments marked by poor parental monitoring and harsh discipline (Farrington & West, 1971; Patterson, 1986; Simons et al., 1994). Thus, antisocial peers may be important environmental influences on both early- and late-onset delinquency with additional negative factors (e.g., poor parental monitoring) coloring the environment of early starters.

Although early literature reviews suggested that delinquency was not as heritable as criminal behavior, a growing twin study literature suggests that childhood antisocial behavior is influenced by genetic factors (Doyle, McGue, & Iacono, 1998; Eaves et al., 1997; Edelbrock et al., 1995; Eley, 1997; Grove et al., 1990; Silberg et al., 1996; Slutske et al., 1997; Taylor, McGue, & Iacono, 2000; see Lyons et al., 1995, for an exception). Of these behavioral genetic studies of antisocial behavior, only three examined differences between early and late occurring antisocial behavior (operationalized as symptoms of conduct disorder [CD] vs. the adult symptoms of antisocial personality disorder [ASPD]). Two studies reported a lower heritability of CD symptoms than adult symptoms of ASPD (Doyle et al., 1998; Lyons et al., 1995). The third study reported a higher heritability of CD symptoms than adult symptoms of ASPD (Grove et al., 1990).

Previous attempts at examining the underlying etiological structure of early- and late-onset antisocial behavior were marked by the use of a single variable to define groups (i.e., ASPD symptoms) and a failure to examine actual age at onset and persistence of

antisocial behavior in the "early-onset" group. As such, it is not clear that earlier studies provided a test of etiological differences between the delinquency subgroups described by Moffitt (1993) and DiLalla and Gottesman (1989).

It may be possible to improve our understanding of the nature of the etiological differences between early and late starters by studying samples that both afford a longitudinal perspective and are genetically informative. Longitudinal data collected on a group of young twins would allow for the careful identification of early starters, late starters, and a nondelinquent comparison group. Such a data set would allow for the examination of phenotypic and environmental differences between early and late starters in relation to the onset of antisocial behavior, and it would also provide a mechanism for directly examining the relative contributions of genetic and environmental influences on subtypes of delinquency.

This investigation was aimed at examining differences in etiological influences on two subtypes of delinquency with an expectation of greater genetic influence for early- than for late-onset delinquency. Early starters, late starters, and nondelinquent controls were carefully selected to test the following hypotheses. First, in part to test the validity of the group selection, we expected early starters to evidence lower verbal intelligence and poorer executive functioning than late starters and controls (who were not expected to differ). Second, based on our contention that early-onset delinquency has an underlying genetic component to its etiology that manifests as problems with inhibition, we expected early starters to evidence more impulsivity, lower constraint, higher negative emotionality, higher rates of ADHD and ODD, and lower SC reactivity (SCR) than late starters and nondelinquent controls (who were not expected to differ). Third, in a direct test of genetic influence on delinquency subtypes, we expected a higher concordance for the early starter phenotype among monozygotic (MZ) twins as compared to dizygotic (DZ) twins, and we expected no substantial difference in concordance among MZ and DZ twins for the late starter phenotype. Moreover, we expected higher rates of antisocial behavior among MZ (as compared to DZ) co-twins of early starters and similar rates of antisocial behavior in co-twins of late starters (regardless of zygosity). Fourth, we expected a higher transmission of antisocial behavior among families of early starters as evidenced by a higher rate of antisocial behavior among (non-co-twin) first- and second-degree relatives of early starters as compared to late starters. Finally, we expected early starters to have a more antisocial peer group than late starters and controls in preadolescence and into adolescence. Given that late-onset delinquency may be largely influenced by peers, we expected an increase in antisocial behavior in the peer group of late starters as they passed into the adolescent risk period for delinquency. We expected nondelinquent controls to have a relatively low level of antisocial behavior in their peer group in preadolescence and adolescence.

Method

Participants

Ten- to 12-year-old boys ($M = 11.32$, $SD = 0.51$) participating in the Minnesota Twin Family Study (MTFS) took part in our experiment. Details about the design of the MTFS are provided in Iacono, Carlson, Taylor, Elkins, and McGue (1999). Briefly, the MTFS is an ongoing, community-

based study of substance abuse and related problems among same-sexed, reared together twins and their parents. Most of the sample (90%) had returned for the first 3-year follow-up assessment (at age 14), and less than half (41%) had returned for the 6-year follow-up (at age 17) at the time of this writing (reflecting the ongoing nature of that data collection wave). Parents of the participating boys had an average of 13.77 (SD = 1.89) years of education and a high school degree (or its equivalent). Consistent with the demographics in Minnesota at the time the twins were born, 98% of the sample was Caucasian.

Zygosity of the twins was established using the following three methods: (a) parents completed a zygosity questionnaire regarding the twins' physical similarity and the frequency with which people confused them, (b) staff rated the zygosity of the twins on the basis of various points of physical similarity (e.g., eye color and ear shape), and (c) zygosity was determined using an algorithm based on ponderal index, cephalic index, and fingerprint ridge count. Any discrepancy in the three methods was resolved through a serological examination of 12 blood group antigens and protein polymorphisms.

The method for placing boys from the initial sample of 766 into three participant groups is outlined in the section *Selection of Participant Groups*. Briefly, 36 (5%) of the boys were identified as *early starters* (boys with a pattern of antisocial behavior problems beginning at or before age 12). Eighty-six (11%) of the boys were identified as *late starters* (boys who had no indication of antisocial behavior until after age 12). A comparison group of 25 *nondelinquent controls* (boys with no signs of antisocial behavior through age 17) was also identified. The use of age 12 as the onset cutpoint was based on previous research (e.g., Farrington et al., 1990; Patterson, Crosby, & Vuchinich, 1992; Tolan & Thomas, 1995).

Measures

Independent Variables

The three participant groups were selected using several indicators of (or precursors to) antisocial behavior. Information from a variety of measures and informants was used in an attempt to minimize problems with phenotypic heterogeneity that may arise when defining groups on a single variable (e.g., symptoms of ASPD) and problems with shared error variance that may arise when single informants are used.

Aggression. At the intake assessment, each boy nominated up to three teachers to complete a teacher rating form (TRF) containing items adapted from the Conners Teacher Rating Scale (Conners, 1969; Pelham, Milich, Murphy, & Murphy, 1989) and the Rutter Child Scale B (Rutter, 1967) addressing (among other things) behavior and peer influences. Six items were rationally selected to create the Aggression Scale, which had acceptable internal consistency reliability ($\alpha = .85$). For each item, a high score indicated a stronger endorsement of the descriptor for the child. The six items were "tough, unforgiving, aggressive," "law abiding" (this item was reverse scored), "has temper outbursts (explosive and unpredictable behavior)," "initiates physical fights," "bullies or intimidates other students," and "is physically cruel to other people." The sum of the items (with no more than one item missing) constituted the Aggression score. All available teacher ratings were averaged for each boy. The mean Aggression scores were transformed (natural log) prior to analysis given the positive skew of the distribution.

Delinquent Behavior Inventory. At intake, each boy completed a self-ratings booklet that contained personality and behavior rating scales. Mothers also rated the boys on some scales. A 36-item delinquent behavior "checklist" adapted from Gibson (1967) was administered as both a self-report and as a mother-report scale. This measure was labeled the *Delinquent Behavior Inventory* (DBI), and it served as an indicator of the variety of delinquent acts committed by the child over his lifetime. Each item was scored 0 (not endorsed) or 1 (endorsed), and the sum of the 36 items constituted the DBI score (calculated separately for mother and child report). The DBI has a unitary factor structure and excellent internal

consistency reliability ($\alpha = .96$). The DBI scales (mother and child reports) were independently transformed ($\log_{10}[x + 1]$) prior to use in the selection process.

CD. Each participant was assessed for a diagnosis of CD using criteria of the revised third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R)*: American Psychiatric Association, 1987) at each MTFS assessment (ages 11, 14, and 17). Symptoms of CD were assessed using structured clinical interviews administered to each boy and his mother independently by trained interviewers (each of whom had at least a BA in psychology). Boys gave information about their CD symptoms at ages 11 and 14 through an MTFS-modified version of the Diagnostic Interview for Children and Adolescents-Revised, Child Version (DICA-R-C; Herjanic & Reich, 1982; Reich & Welner, 1988). Mothers reported on their son's CD symptoms at all three assessments through an MTFS-modified version of the DICA-R, Parent Version (DICA-R-P). At age 17, boys reported on their CD symptoms through an interview created by MTFS staff designed to assess symptoms of ASPD both before and after age 15. Symptoms were assigned by consensus of two or more clinical psychology graduate students using all available clinical information. A computer algorithm generated a best estimate diagnosis on the basis of symptoms endorsed by either informant (e.g., three symptoms plus duration endorsed by at least one informant yielded a CD diagnosis). The reliability of project CD diagnoses generated following this procedure was high ($\kappa = .83$).

Adult antisocial behavior. Symptoms of ASPD assessed at age 17 were tallied for each boy to create adult antisocial behavior (AAB) "diagnoses." Three or more AAB symptoms constituted a "diagnosis" and indicated antisocial behavior problems after age 15 ($\kappa = .95$).

Police contact. The DICA-R-C, DICA-R-P, and a Life Events interview contained a question regarding police contact ("Have you [Have either of the twins] ever been in trouble with the police?"). Age at onset was obtained for positive endorsements. Life Events data were available from each assessment: DICA data were available from the intake assessment.

Selection of Participant Groups

Early Starters

Boys were selected as early starters if at least three of the indicators (described above) from the intake (age 11) assessment were "positive." Indicators from more than one source were treated as single indicators (i.e., a positive indication of police contact at age 11 could be given through the DICA-R-P report, the DICA-R-C report, or the Life Events report). A positive indication on a quantitative variable (e.g., DBI) was defined as a score above one standard deviation from the sample mean (using the log-transformed scale).

Selection for the early starter group required valid information on at least three indicators from the intake assessment. There were multiple configurations of positive indicators used to select early starters. For example, some boys were selected because they had a high aggression rating from teachers, a CD diagnosis by age 12, and a high DBI score. Others were

¹ The threshold of three positive indicators to select early starters was validated, in part, by an analysis of differences in antisocial behavior at ages 14 and 17 that revealed that relaxing the selection threshold to two indicators resulted in the selection of a much larger number of boys as early starters who were substantially less antisocial over time than those selected by the three-indicator threshold. Moreover, the three-indicator threshold selected 5% of the sample as early starters: the expected base rate according to the literature. The two-indicator threshold, on the other hand, identified 18% of the sample as early starters, substantially more than expected from the literature.

selected because they were aggressive in school, had a high DBI score, and had police contact by age 12.

Although we defined our participant groups by age at onset, preliminary analyses indicated that many of our early starters were persistently antisocial. At the time of this report, 89% of the early starters had completed the 3-year follow-up assessment (age 14), at which point 66% received a CD diagnosis and 63% had gotten into trouble with police since their visit at age 11. Of the 28% returning at age 17 (the remaining 72% yet to have a 6-year follow-up scheduled), 40% received an AAB diagnosis and 73% had gotten into trouble with police since their visit at age 14.

Late Starters

Measures at ages 11, 14, and 17 were used to select late starters. The antisocial behavior indicators measured at age 11 were lifetime assessments (with the exception of the teacher rating of aggression, which covered only the period of time the teacher had the boy as a student). Boys were selected as late starters if they lacked any positive indicators of antisocial behavior at age 11 and had at least one positive indicator at age 14 or 17. Moreover, there had to be at least one valid report on each indicator at age 11 (e.g., either mother or child report of the DBI had to be available). Again, there were multiple configurations of positive indicators that qualified boys as late starters. For example, some boys were selected because they had a CD diagnosis at age 14 and no positive indicators at age 11. Other boys were selected because they had police contact (for a nontraffic offense) after age 12 and no positive indicators at age 11.

Nondelinquent Controls

In order to select truly nondelinquent boys as controls, we required valid data on each indicator at each assessment point (to minimize the potential for false negatives). The pool of potential controls was artificially constrained by the fact that only 41% of the 11-year-old cohort had completed their age-17 assessment at the time of this analysis. Boys were selected as controls only if they had no missing data and no positive indicators of antisocial behavior at any age.

Our research question required the identification of relatively homogeneous subgroups of delinquents and a nondelinquent control group. Our careful attention to correct classification led to a large number of the initial sample of 766 boys remaining unselected to a participant group. The majority of the unselected boys (84%) were not selected because of incomplete data on the various indicators of antisocial behavior or too few positive indicators to select as an early starter. A minority (4%) of the unselected boys was missing a piece of data at age 11 precluding selection as a late starter. Finally, 12% of the unselected sample had no indications of antisocial behavior through the 3-year follow-up assessment but were missing data at the 6-year follow-up assessment: thus, their status (late starter vs. control) could not be determined with certainty.

Dependent Variables

Each of the dependent measures, unless noted otherwise, was assessed at the intake visit. The MTFSS assessment is broad and incorporates self-report, interview, and laboratory measures. For various reasons, each dependent variable did not yield data for every case. The most frequent cause of missing data was a failure to properly complete or return self-report forms; data from the laboratory were always missing on participants who completed phone interviews rather than an in-person assessment. All variables (with the exception of the teacher-rated peer measure from the 2 years between study visits) yielded data on 85% (e.g., personality and laboratory measures) to 100% (e.g., psychopathology measures) of the sample. The degrees of freedom for each measure vary somewhat because of missing data. Typically, data were missing on the same proportion of participants in each group.

Cognitive functioning. Estimates of verbal, performance, and full-scale IQ (VIQ, PIQ, and FSIQ, respectively) were obtained from the administration of the Information, Vocabulary, Block Design, and Picture Arrangement subtests of the Wechsler Intelligence Scale for Children-Revised (WISC-R; Wechsler, 1974). At age 14, the boys completed a spatial working memory task that taps prefrontal cortex functioning and thus may serve as a measure of executive functioning (Zald & Iacono, 1998). The delayed memory task required that individuals recall the location of a mark presented on a computer screen following a verbal distraction task. The average error (in distance) was calculated for the 16 trials of the task for each boy.

Personality. The mother's rating booklet contained a scale with 34 descriptors (accompanied by a paragraph explaining the descriptor) designed to tap the broad personality dimensions outlined by Tellegen (1982). The response format ranged from 1 (*my son is definitely low on this trait*) to 4 (*my son is definitely high on this trait*). A principal components analysis yielded a Negative Emotionality (NEM) and a Constraint factor. Items were placed on a scale if they (a) loaded at least .3 on the factor (negatively loaded items were reverse scored) and (b) did not load higher on another factor. The Constraint scale contained 8 items ($\alpha = .73$) related to propensity for thrill-seeking (e.g., "adventurous"), adherence to traditional moral values (e.g., "respecting authority"), and impulsivity (e.g., "planful"). The NEM scale contained 7 items ($\alpha = .69$) pertaining to how the child perceives the world (e.g., "feels exploited"), level of anxiety (e.g., "tense"), and lability of mood (e.g., "even-tempered"). Each scale score was obtained by averaging the responses of the scale items, and higher scores corresponded to higher ratings on the personality dimension.

Impulsivity. Five items from a behavioral control scale in the mother's rating booklet were rationally selected to create the Impulsivity scale ($\alpha = .73$). The scale items referred to uninvited intrusion into games or conversations (2 items), propensity for acting or speaking before thinking (2 items), and ability to deliberately calm down when excited (1 item). Responses were given on a 7-point Likert scale with high values indicating that the behavior strongly described the respondent's son. The Impulsivity score was obtained by averaging the responses to the five scale items; high scores indicated greater impulsivity.

Psychopathology. Lifetime occurrence of symptoms of ADHD and ODD were assessed in the same manner described earlier for CD. However, given the relatively large number of symptoms required for an ADHD or ODD diagnosis under the *DSM-III-R* diagnostic system, it was possible for a boy to meet most of the criteria for the disorder and yet not receive the diagnosis. To account for this possibility, a case was defined as meeting all or all but one of the symptom criteria and duration. Best estimate diagnoses using *DSM-III-R* criteria were assigned using the aforementioned consensus and computer algorithm procedures ($\kappa > .73$).

Psychophysiological variables. The SC data reported here were collected as part of a 3.5hr battery of psychophysiological tests administered to all MTFSS participants. The battery began with an eye-tracking assessment and ended with the recording of central nervous system measures. The autonomic data described here were obtained in the middle of the battery.

Participants were instructed to wash their hands with soap and water; the skin sites were then rubbed with isopropyl alcohol before application of the SC sensors. A 0.79 cm² area of skin was isolated on the fingerprint regions of the index and ring fingers of both hands using electrode collars. Silver-silver chloride electrodes containing a 0.5 molar NaCl electrolyte mixed in Unibase cream (Warner Chilcott Laboratories, Morristown, NJ) were affixed to each of the four fingers, and a constant 0.5 V potential difference was generated across the two fingers on each hand by an SC signal conditioner (Lykken & Venables, 1971). The output from this coupler was recorded through DC amplifiers on a Grass Model 12A Neurodata acquisition system (Grass Instrument Division of Astro-Med., Inc., West Warwick, RI). The SC data were sampled at 256 Hz.

SCR to aversive noise. As the first task in the autonomic assessment, participants were exposed to a single anticipated (but temporally unpredictable) stimulus consisting of a 0.75 s blast of 90 dB white noise presented over headphones while the participant was seated in a reclining chair in a darkened room. The noise blast was presented on the 25th second of a single 40-s trial that began with instructions asking the participant to relax, close his eyes, and wait for a blast of noise. SCR amplitude was scored from each hand and was defined as the difference (in μ siemens) between the 5-s baseline SC level preceding the stimulus and the SC level at the peak of the response curve.

Nonspecific fluctuations in anticipation of an aversive noise. Nonspecific fluctuations (NSFs) were scored on both hands for the 20-s anticipation period preceding the baseline in the task described above. An NSF was defined as a phasic change in SC of at least 0.05 μ siemens between the initial deflection of the curve and the peak of the curve. Only NSFs with a peak during the anticipation period were scored.

Family history of antisocial behavior. A family history interview assessing lifetime symptoms of CD and AAB in each boy's second-degree relatives was administered to parents at intake. Mothers were administered a similar interview assessing lifetime CD and AAB symptoms among the twins' siblings at the 14-year-old assessment. Moreover, the ASPD diagnostic interview (described earlier) was independently administered to biological parents at intake. The aforementioned consensus and computer algorithm procedures were followed to obtain diagnoses of CD and AAB in the parents ($\kappa > .85$). Diagnoses among the boys' siblings and second-degree relatives were obtained from the family history interview. (First-degree relatives included biological parents and nontwin full siblings.)

Antisocial peers. The TRF included items assessing the level of antisocial behavior in each boy's peer group. The TRF was sent to nominated teachers at the 11- and 14-year-old assessments and in each of the 2 years between visits. The Bad Peers scale included four descriptors (tough, dangerous, rebellious, and involved with alcohol and drugs) rated on a 5-point scale with high scores indicating greater applicability of the four descriptors to the boy's peer group ($\alpha = .80$).

Analyses

One-way multivariate analyses of variance (MANOVAs) were applied to quantitative data from conceptually related dependent variables. Significant MANOVAs (using Wilks's lambda) were followed by multivariate contrasts assessing group differences on the pattern of means for all of the dependent variables (see Huberty & Morris, 1989, and Huberty & Smith, 1982, for a discussion of this approach). Nominal variables (e.g., ADHD diagnosis) were examined using the chi-square test. Based on guidelines provided by Cohen (1988), our sample provided adequate power ($\geq 77\%$) to detect medium effects and excellent power ($\geq 90\%$) to detect large effects when alpha was set at .05 for univariate and chi-square tests; our sample provided excellent power ($\geq 98\%$) to detect medium to large multivariate effects (power calculations conducted using GPOWER version 2.0; Faul & Erdfelder, 1992). Missing data did not cause a substantial decrease in power to detect medium or large effects.

The family history variable constituted a family variable in that the information would apply equally to any member of the family. Roughly 40% of each participant group contained concordant twin pairs: 5, 18, and 8 pairs in the control, late starter, and early starter group, respectively. The family history data were identical for both members of the twin pair, and thus one twin from each concordant pair was omitted for the family history analysis (because their data were redundant). Thus, the maximum sample size for that analysis was necessarily reduced from 147 to 116.

A direct examination of differences in genetic and environmental influences on early- and late-onset delinquency was accomplished by calculating probandwise concordance rates for the early and late starter phenotypes. The probandwise concordance rate reflects the risk of affected status to the co-twins of affected individuals and is the preferred concordance

statistic for analysis of categorical data (McGue, 1992). CD symptom counts at ages 11 and 14 among the co-twins of early and late starters were examined to further assess the differences in risk of antisocial behavior among siblings of the same age and sex as the probands.

Results

Our first hypothesis concerned the validity of the group selection; we expected early starters to evidence lower verbal intelligence and poorer executive functioning than late starters and controls (who were not expected to differ). The cognitive functioning data supported our selection of participant groups as the VIQ difference among groups was significant, $F(2, 143) = 5.83$, $p = .004$. Follow-up contrasts revealed that, as expected, early starters ($M = 95.91$) had significantly lower VIQ estimates than controls ($M = 107.16$) and late starters ($M = 102.43$), who did not differ significantly. The groups did not differ significantly in their estimate of FSIQ, $F(2, 143) = 2.26$, or PIQ, $F(2, 143) = 0.05$. Also as expected, early starters evidenced significantly greater error in the delayed memory task than late starters and controls (who did not differ significantly), $F(2, 110) = 6.36$, $p = .002$. Both results are consistent with other reports of cognitive and executive functioning deficits found among persistently antisocial boys (Moffitt, 1993).

Characteristics Related to Inhibition

The second hypothesis was also largely supported by the data. Based on our contention that early-onset delinquency has an underlying genetic component to its etiology that manifests as problems with inhibition, we expected early starters to evidence higher rates of ADHD and ODD, more impulsivity, lower constraint, higher NEM, and lower SCR than late starters and nondelinquent controls (who were not expected to differ significantly).

Psychopathology

The rate of ADHD (± 1 SE) among controls, late starters, and early starters was 0%, 8% (± 3), and 40% (± 8), respectively. The rate of ODD (± 1 SE) among controls, late starters, and early starters was 12% (± 7), 7% (± 3), and 61% (± 8), respectively. As expected, the groups differed significantly ($p < .001$) in their rates of ADHD, $\chi^2(2, N = 147) = 24.62$, and ODD, $\chi^2(2, N = 147) = 46.18$. Follow-up chi-square tests revealed significantly more ODD and ADHD cases among the early starter group than among the controls and late starters (who did not differ significantly).

Personality and Impulsivity

The Impulsivity, Constraint, and NEM data were submitted to a MANOVA and subsequent multivariate contrasts. The expected pattern of personality and impulsivity score differences between early and late starters was found; however, some risk for late-onset delinquency may be conveyed from moderate levels of impulsivity and behavioral undercontrol.

Impulsivity was moderately and significantly ($p < .01$) correlated with Constraint ($r = -.57$) and NEM ($r = .46$); Constraint and NEM were modestly correlated ($r = -.18$, $p = .04$). Table 1 provides descriptive data on each scale for each group. An overall

Table 1
Means and Standard Deviations for the Personality, Impulsivity, and Skin Conductance Measures for the Three Groups

Measure	Control		Late starter		Early starter	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Constraint	3.12	0.37	2.69	0.38	2.24	0.40
NEM	1.99	0.48	1.89	0.38	2.34	0.60
Impulsivity	2.54	0.90	3.09	0.84	3.79	0.93
Log NSF count	.20	.20	.12	.18	.10	.17
Ln SCR	3.84	0.68	4.21	.76	4.32	1.07
NSF count (raw)	.76	.89	.46	.75	.36	.73
SCR (raw)	0.57	0.37	0.89	0.72	1.20	1.02

Note. NEM = negative emotionality; NSF = nonspecific fluctuation; SCR = skin conductance response; log = $\log_{10}(x + 1)$; ln = natural log.

effect for participant group was found for the trait variables, $A = 0.55$, $F(6, 240) = 13.92$, $p < .001$, and was thus followed with multivariate contrasts in which groups were compared for a difference in the pattern or profile of their scores on the three measures. As expected, early starters differed significantly ($p < .001$) from late starters, $\lambda = 0.72$, $F(3, 120) = 15.30$ (canonical correlations for Constraint, NEM, and Impulsivity were .79, -.64, and -.52, respectively), and controls, $\lambda = 0.61$, $F(3, 120) = 25.81$ (canonical correlations for Constraint, NEM, and Impulsivity were -.96, .30, and .58, respectively), on the personality and impulsivity measures. An examination of the group means (in Table 1) revealed that differences were in the expected direction. However, contrary to expectations, the trait variables also differentiated late starters and controls, $A = 0.82$, $F(3, 120) = 8.64$ (canonical correlations for Constraint, NEM, and Impulsivity were .95, .20, and -.52, respectively). The canonical correlations indicated that the Constraint scale contributed most strongly to the differentiation of groups, whereas the NEM scale contributed more to the differentiation of early and late starters than to the differentiation of controls and delinquents.

Psychophysiological Measures

Some boys were missing laboratory data because they completed a phone interview and did not visit, or they reported something during their visit (e.g., hearing loss) that made their data unusable. Determination of the usability of SC data was made without knowledge of diagnostic or participant group status. No lateral differences in SCR and NSF count were found, and thus the data for each measure were averaged across hands. The SCR data were positively skewed and required a natural log transformation. Each raw SCR value was multiplied by 100 prior to taking its natural log in order to avoid an awkward interpretation of the transformed SCR values (i.e., high negative log-transformed values corresponding to small amplitude electrodermal responses). The NSF data were also skewed and were submitted to a $\log_{10}(x + 1)$ transformation prior to analysis. We hypothesized that early starters would show lower autonomic reactivity than late starters and controls; this hypothesis was partly supported by the data.

The correlation between SCR and NSF count was .34 ($p < .01$). A significant relationship between delinquency and electrodermal activity was evidenced in the MANOVA, $\lambda = 0.89$, $F(4,$

244) = 3.45, $p = .009$, and was thus followed by multivariate contrasts. Table 1 contains the descriptive data for the SC measures. Controls differed significantly ($p < .01$) from late starters, $\lambda = 0.93$, $F(2, 122) = 4.84$ (canonical correlations for NSF count and SCR were -.56 and .54, respectively), and early starters, $\lambda = 0.90$, $F(2, 122) = 6.68$ (canonical correlations for NSF count and SCR were -.57 and .53, respectively), with each electrodermal measure contributing about equally to the differentiation between groups. Early and late starters did not differ significantly with regard to their pattern of data on the SC measures, $A = 0.99$, $F(2, 122) = 0.71$. An examination of the means (in Table 1) revealed that differences in NSF count were in the expected direction, whereas differences in SCR were not.

Etiological Influences on Early- and Late-Onset Delinquency

Our two hypotheses regarding differences in genetic influence on early- and late-onset delinquency were also largely supported. In our third hypothesis we expected a higher concordance for the early starter phenotype among MZ twins as compared to DZ twins, and we expected no substantial difference in concordance among MZ and DZ twins for the late starter phenotype. We also expected higher rates of antisocial behavior among MZ (as compared to DZ) co-twins of early starters and similar rates of antisocial behavior among co-twins of late starters (regardless of zygosity). In our fourth hypothesis we expected a higher transmission of antisocial behavior among families of early starters as evidenced by a higher rate of antisocial behavior among first- and second-degree relatives of early starters compared to late starters.

Twin Analysis

Probandwise concordance rates for each phenotype were calculated separately for MZ and DZ twins. This concordance rate reflects the risk of being affected by the phenotype among co-twins of affected individuals with whom they share 100% (MZ) or 50% (DZ) of their genes. The sample included 63 MZ pairs (16 with at least one early starter co-twin; 47 with at least one late starter co-twin) and 33 DZ pairs (12 with at least one early starter co-twin; 21 with at least one late starter co-twin). Consistent with expectations, the risk of being an early starter was substantially greater for co-twins in MZ pairs (55% concordant) than for co-twins in DZ pairs (29% concordant) in which one boy was an early starter. The corresponding tetrachoric correlation for MZ twins was .84 (95% confidence interval (CI): .64, .95) and for DZ twins was .57 (95% CI: .18, .84) yielding a trend (standard normal deviate, $z = 1.55$, $p = .06$, one-tailed) toward a significant difference. For the late starter phenotype, there was relatively little differential risk to co-twins on the basis of level of genetic similarity (MZ and DZ concordance was 43% and 39%, respectively). The corresponding tetrachoric correlation for MZ twins was .64 (95% CI: .46, .79), and for DZ twins it was .57 (95% CI: .27, .80); the difference was nonsignificant, $z = .42$. These data were consistent with our expectation of greater genetic influence on early-onset delinquency than late-onset delinquency. Moreover, 19 (95%) of the 20 pairs of MZ twins who were concordant for delinquency were also concordant for subtype (i.e., early- or late-onset), indicating that when two genetically identical individuals

are concordant for delinquency they almost always have the same form.

Co-Twin Antisocial Behavior

Co-twins of early and late starters were compared on a measure of antisocial behavior at two ages. Figure 1 presents the CD symptom counts (a simple tally of the number of CD symptoms endorsed by the boy or his mother) at two ages for MZ and DZ co-twins of early and late starter probands. The data were submitted to a transformation ($\log_{10}[x + 1]$) prior to analysis. When collapsing across zygosity, co-twins of early starters evidenced significantly more CD symptoms than co-twins of late starters at age 14, $t(64) = -2.34$ ($p = .012$, one-tailed); there was a trend toward a significant difference at age 11, $t(68) = -1.53$ ($p = .065$, one-tailed). Although none of the within-group differences were significant, MZ co-twins of early starters tended to be more antisocial than DZ co-twins, whereas identical and fraternal co-twins of late starters evidenced similar levels of antisocial behavior at ages 11 and 14 (as expected in our third hypothesis).

Family History of Antisocial Behavior

The fourth hypothesis was largely supported by the data as indicated by a greater familial loading for antisocial behavior among early as compared to late starters. Figure 2 presents the percentage of each participant group with (nontwin) first- and second-degree relatives meeting criteria for CD or AAB (or both). The three participant groups did not differ significantly in their total number of first-degree relatives, $\chi^2(2, N = 116) = 3.01$, or second-degree relatives, $\chi^2(2, N = 103) = .35$, and thus we analyzed counts of affected relatives. Nonparametric statistics (Kruskal-Wallis chi-square and Mann-Whitney follow-up tests) were used to evaluate the data given its substantial skew. The groups differed significantly in the number of first-degree relatives with AAB, $\chi^2(2, N = 116) = 16.86$, $p < .001$, but not CD, $\chi^2(2, N = 116) = 0.61$. Early starters had significantly more first-degree

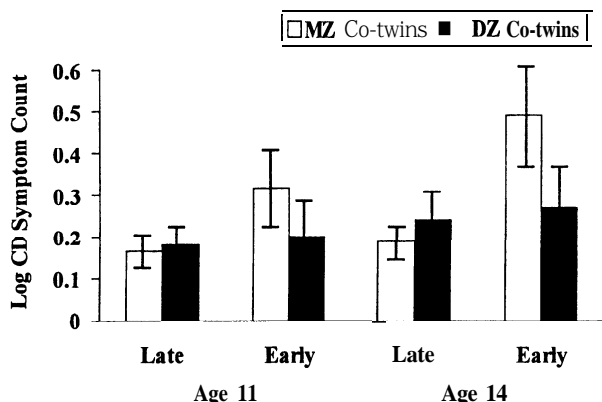


Figure 1. Mean conduct disorder symptom count among co-twins of early and late starters at ages 11 and 14. CD = conduct disorder; Log = $\log_{10}(x + 1)$; MZ = monozygotic; DZ = dizygotic; error bars represent ± 1 SE. Late starters: MZ = 34 co-twins ($n = 33$ at age 14); DZ = 16 co-twins ($n = 15$ at age 14); early starters: MZ = 10 co-twins; DZ = 10 co-twins ($n = 8$ at age 14).

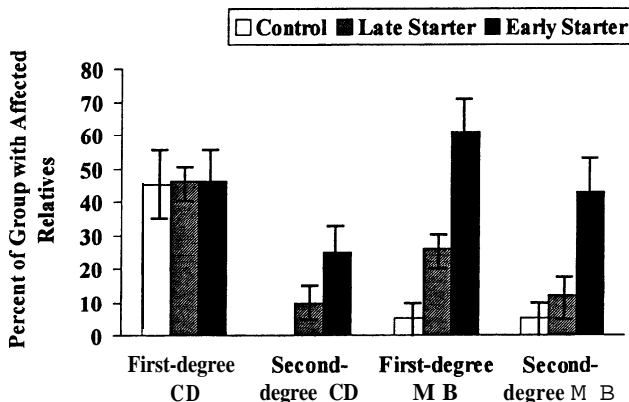


Figure 2. Percentage of each group with antisocial behavior problems among first- and second-degree relatives. CD = conduct disorder; AAB = adult antisocial behavior; error bars represent ± 1 SE.

relatives with AAB than late starters and controls (who did not differ significantly). The groups differed significantly in the number of second-degree relatives with AAB, $\chi^2(2, N = 115) = 8.35$, $p = .015$, and CD, $\chi^2(2, N = 115) = 6.05$, $p = .05$. Early starters had significantly more second-degree relatives with AAB or CD than controls and significantly more second-degree relatives with AAB (but not CD) than late starters. Late starters and controls had similar family histories of antisocial behavior.

Peer Influence

The data supported our fifth hypothesis that early starters have a more antisocial peer group than late starters and controls in preadolescence and adolescence. The data provided some support for our expectation of an increase in antisocial behavior in the peers of late starters prior to adolescence.

Figure 3 depicts the change in Bad Peers scores for each group over 3 years. The Bad Peers scale distributions at each assessment point were positively skewed, and each was submitted to a natural log transformation prior to analyses. A repeated measures analysis of variance (ANOVA) was used to assess main effects for group and time of assessment as well as the interaction between those two effects. There was a nonsignificant trend for a main effect for time of assessment, $F(2, 134) = 2.99$, $p = .054$. The interaction between time of assessment and participant group was nonsignificant, $F(4, 134) = 1.65$. The main effect for group was significant, $F(2, 67) = 14.27$, $p < .001$. Univariate tests indicated that, as expected, early starters had a significantly more antisocial peer group than both late starters (who had not yet onset in their antisocial behavior; $d = 1.41$) and controls ($d = 1.64$), $F(2, 122) = 33.77$, $p < .001$. A significant ($p < .001$) effect also was found at the follow-up interval, $F(2, 99) = 14.14$, and at the 14-year-old assessment, $F(2, 106) = 10.44$. Again, early starters had a significantly more antisocial peer group than the late starters ($d = .85$ for the follow-up interval and $.60$ at age 14) and controls ($d = 1.56$ for the follow-up interval and 1.22 at age 14). As expected, late starters had a significantly more antisocial peer group than controls during the follow-up interval ($d = .72$) and at age 14 ($d = .62$). The data were consistent with the notion that

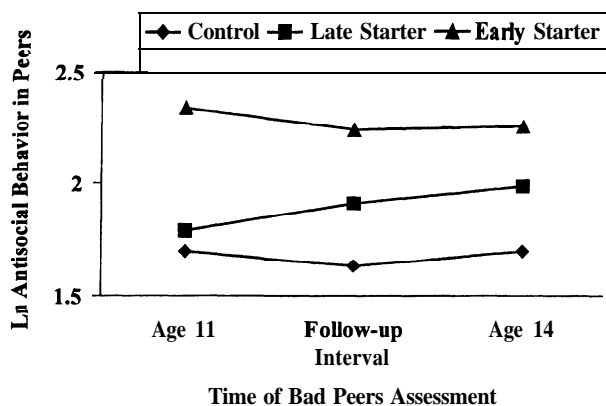


Figure 3. Mean teacher rating of antisocial behavior in peer group of early starters, late starters, and nondelinquent controls. Ln = natural log. The follow-up interval refers to the 2-year period between assessment visits; scores for the follow-up interval represent the average of the Bad Peers scores from each of the two visit interval years. At age 11, 24 nondelinquent controls, 71 late starters, and 30 early starters were assessed; at follow-up, 23 controls, 64 late starters, and 15 early starters were assessed; and at age 14, 23 controls, 63 late starters, and 23 early starters were assessed.

negative peer influences increase among late starters prior to the onset of their antisocial behavior.

Discussion

The aim of this study was to examine differences in genetic and environmental influences on two subtypes of delinquency. One advantage of this investigation over previous studies examining genetic and environmental influences on early and late occurring antisocial behavior was the use of a unique longitudinal, genetically informative data set. This data set allowed us to identify twins as early starters, late starters, and nondelinquent controls by combining information from various sources (self-reports, diagnostic interviews), informants (teachers, parents, children), and assessments covering the risk period for early starters (i.e., at or prior to age 12) as well as most of the adolescent risk period for late-onset delinquency (ages 13–17). Moreover, we used patterns of various indicators of antisocial behavior to define the participant groups rather than relying on a single variable, which may have helped reduce the heterogeneity of the participant groups in the present study. Finally, the longitudinal design allowed us to show that the early starters were persistently antisocial (as predicted by developmental theories).

The hypotheses in this study were largely supported by the data. Briefly, as compared to late starters and nondelinquent controls, early starters had lower verbal and spatial memory functioning, more problems related to psychological, emotional, and behavioral inhibition, higher negative emotionality, earlier and more persistent association with antisocial peers, and higher familial transmission of antisocial behavior and greater genetic influence on their phenotype.

Although our data suggested a greater genetic influence on early-onset than late-onset delinquency, a larger sample would be needed to rule out the possibility of genetic influence on late-onset

delinquency. Our data are consistent with the behavioral genetic literature on antisocial behavior across the life span as outlined by DiLalla and Gottesman (1989), namely, that early starters (who are expected to show antisocial behavior into adulthood) have substantial genetic effects on their antisocial behavior whereas late starters (who are expected to have a transitory course of delinquency) have perhaps only modest genetic effects on their antisocial behavior.

What does the genetic influence on early-onset, persistent antisocial behavior translate into in terms of traits or behaviors? The data from this study were consistent with our proposal that the genetic influence represents an underlying biological liability toward disinhibition. That is, early starters possess behavioral and personality problems related to inhibition that increases the likelihood that they exhibit antisocial behavior at an early age (because many of these characteristics develop early) and over the course of the life span (because many of these characteristics are fairly stable). Our data on personality and impulsivity also indicated that antisocial behavior (in general) may be related to individual differences in disinhibition given that late starters appeared less inhibited than nondelinquents prior to the late starters' onset of delinquency (according to the multivariate contrasts for the trait measures).

The genetic influence on antisocial behavior may manifest as deviance in inhibition. However, according to the literature, unique environmental experiences have a larger impact on individual differences in delinquency than genetic (or shared environmental) factors. Our data on peers was consistent with the notions put forth by Harris (1998) and others who suggested that peers (typically viewed as a nonshared environmental influence) are salient socializing agents that determine behavioral and perhaps psychological characteristics or outcomes. The presence of antisocial behavior in our participant groups appeared to be fairly closely tied to the presence of antisocial behavior in the peer group. However, our data were not suited to determine the reason for this apparent association. Do early starters associate with antisocial peers because of rejection by nondelinquent peers, as Patterson (1986) suggested? Or do early starters select as friends those individuals who are more deviant (like them) in terms of personality, behavior problems, intellectual ability or performance in school, and other genetically mediated traits? Multivariate behavioral genetic models may help address these questions.

This study had some clear advantages over previous investigations; however, there were also some important limitations. First, our study was limited to an examination of subgroups of delinquent boys. It is unclear how well models of delinquency that are being constructed from data on boys map onto data from girls. Future work should attempt to include (or perhaps focus on) subgroups of delinquent girls. Second, although we used great care to select nondelinquent controls, we recognize that some of the boys we identified for that group may exhibit antisocial behavior (of the late-onset variety) by the time of their next study visit at around age 20. Third, our sample was Caucasian and thus our results should be applied cautiously to other ethnic groups. Finally and perhaps most important, our sample had relatively low power to directly test for differences in genetic influence on subtypes of delinquency. Rather, our conclusions were based on the converging of evidence from a variety of tests examining the issue from various angles. Moreover, our sample was quite limited in its

power to test for small effects, despite its adequate power to test for medium to large effects between groups. As such, larger samples of early starters, late starters, and nondelinquent controls would be needed to detect any subtle differences between groups.

Like any novel finding, our results await replication and expansion. Specifically, our findings raise questions about the possible correlation among the genetic factors associated with variance in antisocial behavior, peer influence, and personality and behavior problems related to inhibition; about the effects of phenotypic heterogeneity on heritability estimates for delinquency; about the developmental timing of the emergence of antisocial behavior, personality traits, psychopathology, and peer group influences; and about other possible indices of disinhibition that differentiate subtypes of delinquents. The present study highlights the value of trying to understand antisocial behavior through the comprehensive study of individual differences in the phenotype.

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