Prediction of Differential Adult Health Burden by Conduct Problem Subtypes in Males

Candice L. Odgers, PhD; Avshalom Caspi, PhD; Jonathan M. Broadbent, BDS; Nigel Dickson, MD; Robert J. Hancox, MD; HonaLee Harrington, BS; Richie Poulton, PhD; Malcolm R. Sears, MD; W. Murray Thomson, PhD; Terrie E. Moffitt, PhD

Context: A cardinal feature of the *DSM-IV* diagnostic criteria for conduct disorder is the distinction between childhood- vs adolescent-onset subtypes. Whether such developmental subtypes exist in the population and have different prognoses should be rigorously tested to inform the *DSM-V*.

Objectives: To evaluate the epidemiological validity of childhood- vs adolescent-onset conduct problems in a prospective birth cohort, and to assess whether life-course-persistent conduct problems are associated with a greater adult health burden.

Design, Setting, and Participants: Our sample includes 526 male study members in the Dunedin Multidisciplinary Health and Development Study, a 1-year birth cohort (April 1, 1972, through March 30, 1973). Developmental trajectories were defined using prospective ratings of conduct problems at 7, 9, 11, 13, 15, 18, 21, and 26 years of age.

Main Outcome Measures: Health burden was assessed as mental and physical health problems at 32 years of age measured via diagnostic interviews and physical examinations.

Results: We identified the following 4 developmental subtypes of conduct problems through general growth mixture modeling: (1) childhood-onset/life-course-persistent, (2) adolescent onset, (3) childhood limited, and (4) low. At 32 years of age, study members with the life-course-persistent subtype experienced the worst health burden. To a lesser extent, those with the adolescent-onset subtype also experienced health problems. A childhood-limited subtype not specified by *DSM-IV* was revealed; its adult health outcomes were within the range of the cohort norm.

Conclusions: Results support the epidemiological validity of the *DSM-IV* conduct disorder distinction based on age of onset but highlight the need to also consider long-term persistence to refine diagnosis. Preventing and treating conduct problems has the potential to reduce the adult health burden.

Arch Gen Psychiatry. 2007;64:476-484

Author Affiliations: Social, Genetic, and Developmental Psychiatry Centre, Institute of Psychiatry, King's College London, London, England (Drs Odgers, Caspi, and Moffitt); Departments of Psychology and Neuroscience, and Psychiatry and Behavioral Sciences, and Institute for Genome Sciences and Policy, Duke University, Durham, NC (Drs Caspi and Moffitt); Department of Psychology, University of Wisconsin, Madison (Ms Harrington); Department of Oral Sciences and Orthodontics, School of Dentistry (Drs Broadbent and Thomson), and Dunedin Multidisciplinary Health and Development Research Unit (Drs Hancox and Poulton), Department of Preventive & Social Medicine (Dr Dickson), Dunedin School of Medicine, University of Otago, Dunedin, New Zealand; and Department of Medicine, McMaster University, and Firestone Institute for Respiratory Health, Hamilton, Ontario (Dr Sears).

SINCE LEE ROBINS' SEMINAL 1966 publication, *Deviant Children Grown Up*,[1] it has been known that conduct disorder (CD) in childhood predicts negative life outcomes. Longitudinal research has consistently identified childhood-onset conduct problems as one of the most robust predictors of later involvement in serious offending behavior.[2-4] The unique risks that accompany early-onset conduct problems have been recognized in the distinction between childhood- and adolescent-onset subtypes of CD made by the *DSM-IV*.[5] During the past decade, research generated by a developmental taxonomy of antisocial behavior[6] has refined this distinction by identifying a relatively small but persistent and pathological subgroup of male individuals who demonstrate conduct problems that begin in childhood *and* persist into adulthood; this subgroup, classified as life course persistent (LCP), has been shown to have unique neurodevelopmental origins that are exacerbated by high-risk social and familial contexts.[7] Like the *DSM-IV*, the taxonomy contrasts the LCP subtype against a more common and transient subtype, classified as adolescent limited, whose antisocial behaviors emerge during adolescence but are expected to desist once they reach adulthood.

The fundamental divide between childhood- vs adolescent-onset CD has guided research and policy debates and become a dominant heuristic within clinical practice.[8,9] The validity of this distinction, however, has not been fully tested and should be delineated before the *DSM-V* is written.[10] At least 2 important questions remain unanswered: first, whether childhood- and adolescent-onset subtypes can be identified within the population (epidemiological validity) and, second, whether these subtypes have different adult prognoses (predictive validity). The present study benefits from state-of-the-art techniques for the analysis of longitudinal data[11] to test whether these subgroups can be recovered from a 30-year epidemiological cohort. This research also expands previous work by testing whether predictive validity can be extended to encompass health outcomes in adulthood. Specifically, we ask whether individuals in

the LCP class have the highest rates of mental and physical health problems by 32 years of age. To our knowledge, this is the first study to test the epidemiological and predictive validity of the childhood- vs adolescent-onset distinction within a population cohort.

TESTING EPIDEMIOLOGICAL VALIDITY

The application of general growth mixture modeling (GGMM)[12-14] allowed us to assess whether the childhood- and adolescent-onset subgroups could be identified within our population cohort. The GGMM procedure identifies the ideal number of classes that underlie a population distribution and provides parameters that characterize each class. Four criteria are used to query whether the hypothesized classes are present. First, does the model that demonstrates the best empirical fit also contain the expected number of classes? Based on the *DSM-IV*, the following 3 classes are expected: childhood onset, adolescence onset, and normative (low). Second, does the shape of each class map on to the developmental taxonomy? The childhood-onset (LCP) class is expected to have high levels of conduct problems beginning in childhood and persisting to adulthood. The adolescent-onset (or adolescent-limited) class is predicted to initiate conduct problems in midadolescence and desist in adulthood, whereas the low class is expected to remain low across development. Third, are the estimated prevalence rates of each class comparable to the rates implied by the taxonomy? The LCP class is expected to be relatively small (5%-10%), the adolescent-onset class should be more common, and the low class should constitute most of the population.[7] Fourth, the solution is externally validated by asking whether the profile of childhood risk in the LCP class is differentially characterized by social, familial, and neurodevelopmental deficits, whereas the profile of the adolescent-onset class is not.

TESTING PREDICTIVE VALIDITY

The utility of the distinction between childhood- and adolescent-onset forms of conduct problems can also be evaluated in terms of predictive validity. Research to date has consistently linked the LCP pathway to continued involvement in antisocial and criminal acts.[7,15] However, adult prognosis has been hypothesized to extend beyond crime to encompass mental and physical health problems.[16] If this prediction holds, the cumulative burden of disease for individuals in the LCP class may be greater than assumed. The design of this study allowed us to link conduct problem subtypes among study members with their adult health burden, herein assessed according to the presence of mental health problems, physical health problems, and victimization of others.

METHODS

PARTICIPANTS

Participants are male members of the Dunedin Multidisciplinary Health and Development Study, and study protocols were approved by the institutional review boards of the participating universities. The cohort of 1037 children (51.6% male) was constituted at 3 years of age, when investigators enrolled 91.0% of consecutive births from April 1, 1972, through March 30, 1973, in Dunedin, New Zealand. Cohort families represent the full range of socioeconomic status in New Zealand's South Island and are primarily white. Follow-up assessments were conducted with informed consent at 5, 7, 9, 11, 13, 15, 18, 21, 26, and 32 years of age, when 95.8% of the living study members underwent assessment in 2003 to 2005. Cross-national comparisons lend confidence regarding the generalization of findings from the Dunedin Study population to other industrialized nations.[17]

MEASURES

Conduct problems were measured at 7, 9, 11, 13, 15, 18, 21, and 26 years of age through scoring the following 6 key symptoms of CD as being present or absent at each age: physical fighting, bullying others, destroying property, telling lies, truancy, and stealing. A composite score, ranging from 0 to 6, was formed representing antisocial behavior during the past year. Other CD symptoms were not used because they did not cover the study's age span (eg, running away and staying out late) or had very rare prevalence (eg, fire setting, forced sex, and animal cruelty). Each of the 6 conduct problem symptoms was operationalized through multiple items collected at each age. Item content was adapted across the age span to ensure that the measures were developmentally appropriate. For example, truancy included items such as skipping school for younger students and work absenteeism for older, employed study members; bullying included items such as bullying other children, threatening violence, and, at older ages, robbery; and stealing included items such as stealing from school or home, shoplifting, auto theft, absconding from a rental with unpaid bills or rent, and embezzlement from employers. The study's reporting sources were also developmentally appropriate, including parents and teachers in childhood; self, parents, and teachers in adolescence; and self alone in adulthood. Further details are presented elsewhere.[17] The developmental appropriateness of the scale was supported by multiple-group confirmatory factor analysis in which equality constraints on the factor loadings across the ages of 7 to 26 years did not significantly reduce the model fit ($\Delta\chi^2 = 25.1/\Delta df = 20$; $P = .20$).

CHILDHOOD PREDICTORS

Parent criminal conviction was measured by parental report in 1998, when the parents' ages ranged from 40 to 75 years. Of parents, 13.2% reported they had been convicted of a crime.[17]

Socioeconomic status was measured as the highest of the father's or the mother's occupation using a 6-point scale for New Zealand[18]; 20.9% of the families were classified as having low socioeconomic status.

Maltreatment was measured using staff observations of rejecting mother-child interaction at 3 years of age, parental reports of harsh discipline at 7 and 9 years of age, 2 or more changes in primary caregivers to 11 years of age, and retrospective reports by study members at 26 years of age of injurious physical abuse or unwanted sexual contact before 11 years of age. In all, 9.2% of the sample had 2 or more indicators of maltreatment.[19]

The mother's IQ was tested using the Science Research Associates Verbal Form[20] when the children were 3 years of age (score standardized to population mean, 100; SD, 15). A low IQ in the mother was defined as less than 85 on the standardized score.

The child's IQ was tested using the Wechsler Intelligence Scale for Children–Revised[21] at 7, 9, 11, and 13 years of age, and the 4 values were averaged to enhance reliability (score stan-

dardized to population mean, 100; SD, 15).[22] A low IQ in the child was defined as less than 85 on the standardized score.

Undercontrolled temperament was measured through staff ratings after observing the child in a 90-minute testing session with an unfamiliar examiner at 3 years of age. Factor and cluster analyses reduced these ratings to 3 temperament types, including the undercontrolled type,[23] since replicated in other samples.[24-26]

Attention-deficit/hyperactivity disorder was measured using the Diagnostic Interview for Children–Child Version[27] at 11, 13, and 15 years of age. Diagnoses were made according to the *DSM-III*[28] and confirmed through parent or teacher report[17]: 9.6% of the male children met diagnostic criteria for attention-deficit/hyperactivity disorder.

MENTAL HEALTH OUTCOMES AT 32 YEARS OF AGE

Psychiatric disorders during the past year at 32 years of age were assessed in private structured interviews using the Diagnostic Interview Schedule for *DSM-IV*.[29] Diagnoses were made according to the *DSM-IV* criteria.[5] Prevalence rates in the Dunedin cohort are similar to those from American epidemiological surveys.[30,31] For this report, we examined grouped anxiety disorders (generalized anxiety disorder, obsessive-compulsive disorder, panic disorder, agoraphobia, social phobia, and simple phobia), major depressive disorder, cannabis dependence, dependence on other drugs, alcohol dependence, and posttraumatic stress disorder.

Indicators of mental health impairment in the 6 years from 26 to 32 years of age were measured using a life history calendar.[32,33] Study members reported periods when they received outpatient treatment for mental health or substance abuse problems, periods when they took psychiatric medication, all psychiatric inpatient hospitalizations and suicide attempts, and periods when they were homeless or were taken in by friends or relatives because they had no place to live.

Partner abuse in the past year at 32 years of age was measured in a standardized interview about 13 physical abuse acts (eg, slapping, strangling, kicking, hitting, beating up, forcing sex, and using a weapon) and 13 controlling abuse acts (eg, damaging their partner's clothes, car, or pet; stopping their partner from contacting family or friends; and stalking). Results are presented for the 87.1% of men who were in a relationship or who had dated in the past year. The men's self-reports have been previously validated against their partners' reports.[34]

Self-reported violence in the past year at 32 years of age was measured using the US National Youth Survey Self-report Crime Interview.[35] Items ascertained simple assault, aggravated assault, gang fighting, robbery, arson, and forced sex.[36] Assaults against partners were excluded to avoid overlap with the partner abuse measure.

Conviction for violent crimes (official) from 26 to 32 years of age was measured by searching the computerized New Zealand police database. Convictions included but were not limited to common assault, common domestic assault, assault of a child, assault with a weapon, rape, indecent assault on a female, robbery aggravated with a firearm, assault of a female subject by a male subject with a weapon, resisting police, and arson.

PHYSICAL HEALTH OUTCOMES AT 32 YEARS OF AGE

Self-reports of Health

Study members provided reports of their overall health on a 5-point scale (1 indicates poor; 5, excellent).[37] They also reported how often they received treatment for physical health problems from a general practitioner and whether they had been hospitalized in the past year.

Cardiovascular Disease Risk

Because the cohort is still too young to present clinical end points of cardiovascular disease (eg, myocardial infarction), we focused on multiple risk-factor clustering as a measure of cardiovascular risk.[38,39] Six biomarkers were used: overweight, high blood pressure, elevated total cholesterol level, low high-density cholesterol level, elevated glycosylated hemoglobin level, and low maximum oxygen consumption. Study members were clustered if they had at least 3 risk factors.[40]

High-Sensitivity C-Reactive Protein

High-sensitivity C-reactive protein level is thought to be one of the most reliable measured indicators of vascular inflammation[41] and has been recently endorsed as an adjunct to traditional risk factor screening for cardiovascular risk.[41,42] Individuals with high-sensitivity C-reactive protein levels higher than 3.0 mg/L were considered to be at high risk.[41]

Respiratory Function

Respiratory function was assessed with the use of a computerized spirometer and body plethysmograph; technical details are provided elsewhere.[43] Measurements of vital capacity were repeated to obtain at least 3 repeatable values (within 5%), followed by full forced expiratory maneuvers to record forced expiratory volume in 1 second. The ratio of the forced expiratory volume in 1 second to vital capacity is reported as the primary lung function measure because it is the most sensitive measure for assessing airway remodeling in a large cohort.[44] Study members self-reported symptoms of chronic bronchitis[45] as chronic coughing and phlegm. Smoking during the past year was assessed as part of the Diagnostic Interview Schedule.[29] Tobacco dependence was diagnosed according to *DSM-IV* criteria.[5]

SEXUAL HEALTH

Serology for herpesvirus 2 infection was performed using an indirect enzyme-linked immunosorbent assay (HerpeSelect 2 ELISA IgG; Focus Technologies, Cypress, Calif).[46] Herpesvirus 2 infection was diagnosed using a cutoff value of 3.5, and any equivocal result (from 0.9 to 3.5) was resolved using herpesvirus 2 Western blot analysis.[47]

DENTAL HEALTH

Examinations were conducted using calibrated dental examiners in all 4 quadrants; technical procedures are described elsewhere.[48] We report the number of untreated decayed surfaces present at 32 years of age and the presence of gum disease (defined as $\geq$2 sites with $\geq$4 mm of combined attachment loss).

INJURIES

Study members reported serious injuries between 26 and 32 years of age, defined as any injury requiring treatment from a physician, a medical center, or emergency services. We report the percentage who experienced an injury and, among these individuals, the percentage with a non–sports-related injury.

STATISTICAL ANALYSES

Analyses proceeded in 3 steps. First, we used GGMM to identify subclasses of individuals on the basis of distinct profiles of conduct problems from 7 to 26 years of age. The GGMM procedure

Table 1. Fit Indices for GGMM Solutions Among 526 Males in the Dunedin Multidisciplinary Health and Development Study

No. of Latent Classes	BIC*	AIC*	Entropy†	LMR-LRT‡
1	13 990.39	13 978.20	. . .	. . .
2	13 308.39	13 292.00	0.81	.008
3	13 126.77	13 106.00	0.81	.01
4	12 930.00	12 905.00	0.80	<.001
5	12 915.80	12 886.41	0.77	.43
6	12 877.00	12 843.20	0.76	.89

Abbreviations: AIC, Akaike information criterion; BIC, Bayesian information criterion; GGMM, general growth mixture modeling; LMR-LRT, Lo-Mendell-Rubin likelihood ratio test; ellipses, cannot be calculated.
*Balances model complexity and goodness of fit to the sample data, with smaller values representing a better fit.
†Entropy refers to the average classification accuracy when assigning participants to trajectory classes, with values closer to 1 indicating greater precision.
‡Provides a direct test between 2 models. A low *P* value indicates that a *k* – 1 class model should be rejected in favor of a model with at least *k* classes.

is an extension of latent growth curve modeling[49,50] in which key parameters of the growth process are allowed to vary by trajectory class. In traditional latent growth curve modelling, Y is an observed variable assessed over multiple occasions (t=1 to T) on some persons (n=1 to N) and can be represented as $Y[t]_n = y_{0n} + A[t]y_{sn} + e[t]_n$; where y_0 represents an individual's initial level of conduct problems; y_s represents an individual's change over time; $A[t]$ represents the basis weights that define the shape of change over time for the group; and $e[t]$ represents measurement error at each occasion. General growth mixture modeling differs from traditional latent growth curve modeling in that it does not assume that individuals are drawn from a single population and instead allows subclasses of individuals to vary around different mean growth curves.[14] The GGMM procedure has been widely applied in developmental research. The technical details of GGMM are described elsewhere.[51]

Models were fitted in Mplus version 3.13[52] using maximum likelihood estimation. Classification quality was assessed through recommended indices, including the Bayesian information criterion,[53] Akaike information criterion,[54] Lo-Mendell-Rubin likelihood ratio test,[55] and entropy.[14] Missing data were handled through full information maximum likelihood[56]; 98.1% of the sample had 3 or more observations and 81.7% had complete data for at least 7 of 8 observations. Individuals were assigned to their most likely class based on posterior probabilities. Second, as one means of validating the solution, we conducted standard between-group tests (eg, χ^2 test and analysis of variance) using childhood covariates previously hypothesized to differentiate the classes. Third, to evaluate the predictive validity of the GGMM solution, we compared classes on health outcomes at 32 years of age.

RESULTS

A 4-class model was selected as the best model on the basis of empirical fit indices (Bayesian information criterion, 12 930.0; Akaike information criterion, 12 905.0; entropy, 0.80; Lo-Mendell-Rubin likelihood ratio test, $P<.001$) and correspondence with a priori expectations derived from the *DSM-IV* and the developmental taxonomy. As demonstrated in **Table 1**, the decrease in the Bayesian information criterion (13 126.8 to 12 930.0) and Akaike information criterion (13 106.0 to 12 905.0) between a 3- and a 4-class model was large, and the Lo-Mendell-Rubin likelihood ratio test favored rejecting the 3-class model ($P<.001$). Moving from a 4- to a 5-class model, however, was not well supported; the relative change in the Bayesian information criterion (12 930.0 to 12 915.8) and Akaike information criterion (12 905.0 to 12 886.4) was small, classification quality was lower (entropy, 0.77), and the Lo-Mendell-Rubin likelihood ratio test favored rejecting the 5-class model ($P=.43$). The trajectories included an LCP class (10.5% of the cohort) who initiated antisocial behavior in childhood with persistence into adulthood, an adolescent-onset class (19.6%) whose conduct problems emerged during adolescence, a childhood-limited class who demonstrated conduct problems in childhood but subsequently desisted (24.3%), and a low class (45.6%) characterized by low levels of conduct problems (**Figure**).

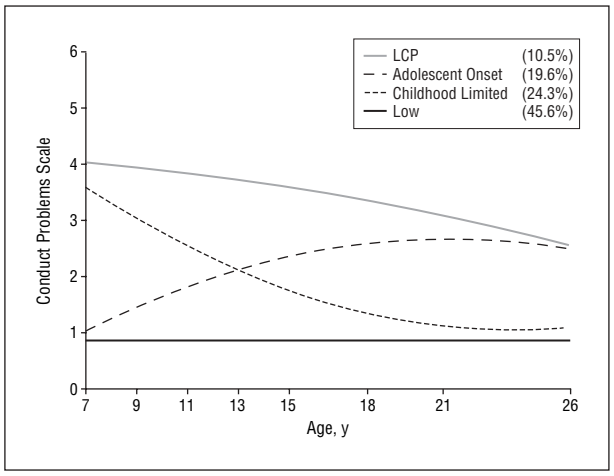

Figure. Conduct problem trajectories from 7 to 26 years of age in a 4-class model of 526 male study members. LCP indicates life course persistent.

Table 2 presents the prevalence of childhood risk factors by trajectory-class membership. Results from this table highlight 3 main findings. First, the LCP class had the most compromised childhood histories as indexed by measures of social, familial, and neurodevelopmental deficits. The LCP class differed significantly from the other 3 classes on measures of parental criminality, socioeconomic status, and childhood diagnosis of attention-deficit/hyperactivity disorder (columns A, B, and C). The LCP class also differed significantly from the low and adolescent-onset classes on childhood measures of maltreatment and IQ (columns A and C). Second, the adolescent-onset class bore the closest resemblance to the low class on childhood risk factors, with no statistically significant differences between the adolescent-onset and low classes on measures

Table 2. Prevalence Rates of Childhood Risk Factors by Trajectory-Class Membership Among Males in the Dunedin Multidisciplinary Health and Development Study

	Trajectory Class, % of Subjects				Class Comparison, OR (95% CI)				
Childhood Predictors (n)	Low	CL	AO	LCP	A. LCP vs Low	B. LCP vs CL	C. LCP vs AO	D. AO vs Low	E. CL vs Low
Parent convicted (479)	11.7	9.6	12.4	31.1	3.4 (1.6-7.3)	4.2 (1.7-10.3)	3.2 (1.3-7.7)	1.1 (0.5-2.2)	0.8 (0.4-1.7)
Low SES (523)	13.4	21.9	23.5	40.7	4.5 (2.3-8.6)	2.5 (1.2-4.9)	2.2 (1.1-4.5)	2.0 (1.1-3.6)	1.8 (1.03-3.2)
Maltreatment (526)	2.1	12.5	7.8	23.6	14.5 (4.9-42.9)	2.2 (1.0-4.9)	3.7 (1.4-9.5)	4.0 (1.3-12.4)	6.7 (2.4-18.8)
Mother's IQ low (509)	14.5	23.0	16.2	29.6	2.5 (1.2-4.9)	1.4 (0.7-2.9)	2.2 (1.0-4.8)	1.1 (0.6-2.2)	1.8 (1.01-3.1)
Child's IQ low (512)	6.9	22.8	14.9	30.2	5.8 (2.7-12.6)	1.5 (0.7-3.0)	2.5 (1.1-5.5)	2.3 (1.1-4.9)	4.0 (2.1-7.7)
Undercontrolled temperament (519)	5.5	21.3	15.7	17.0	3.5 (1.4-8.8)	0.8 (0.3-1.7)	1.1 (0.5-2.7)	3.2 (1.5-6.9)	4.6 (2.3-9.4)
ADHD diagnosis (498)	3.1	12.3	6.0	37.7	18.7 (7.3-47.7)	4.3 (2.0-9.4)	9.5 (3.5-25.7)	2.0 (0.6-6.0)	4.3 (1.7-10.9)

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; AO, adolescent onset; CI, confidence interval; CL, childhood limited; LCP, life course persistent; OR, odds ratio; SES, socioeconomic status.

of parental criminality, the mother's IQ, and attention-deficit/hyperactivity disorder (column D). Third, conduct problems during childhood were associated with multiple childhood risk factors regardless of whether the problems persisted over time. That is, the childhood-limited class differed significantly from the low class on virtually every measure (6 of 7) (column E).

Table 3 presents mental health outcomes at 32 years of age by trajectory-class membership. Results from Table 3 convey 3 main findings. First, the LCP class generally had the worst mental health outcomes. Differences between the LCP and low classes reached statistical significance for 13 of 15 outcomes (column A) and differences between the LCP and childhood-limited classes were found for 11 of the 15 outcomes (column B). The LCP class also had worse mental health problems than did the adolescent-onset class, with differences reaching statistical significance for anxiety, major depressive disorder, other drug dependency, homelessness, and violence convictions (column C).

The second main finding conveyed in Table 3 is that the adolescent-onset class also experienced mental health problems at 32 years of age. Compared with the low class, the adolescent-onset class was significantly elevated on 9 of 15 mental health outcomes (column D). In particular, the adolescent-onset class was more likely than the low class to have a substance use disorder, to have received services for mental health problems (including treatment, medication, and hospitalization), and to have victimized others.

The third main finding conveyed in Table 3 is that the childhood-limited class was doing relatively well, having significantly higher prevalence than the low class for only 2 outcomes (column E).

To summarize the results in Table 3, we created 3 summary variables that indexed the numbers of psychiatric disorders, mental health impairments, and types of victimization of others for each study member. The means and corresponding effect sizes (Cohen d) by trajectory class are displayed at the bottom of Table 3. In general, moderate to large effect sizes were found when the LCP class was compared with all other classes (range of d, 1.01-1.18 vs low class; 0.61-0.82 vs childhood-limited class; and 0.29-0.57 vs adolescent-onset class). Moderate effect sizes were found between the adolescent-onset and low classes (range of d, 0.40-0.77), with small effect sizes reported between the childhood-limited and low classes (range of d, 0.22-0.27).

Table 4 presents physical health outcomes at 32 years of age by trajectory-class membership. Similar to the pattern of mental health outcomes, 3 main findings are conveyed by this table. First, to varying degrees, the LCP class had the highest rates of physical health problems. Differences between the LCP and low classes reached statistical significance on 12 of 14 outcomes (column A), and differences between the LCP and childhood-limited classes were found on 8 of 14 outcomes (column B). When compared with the adolescent-onset class, the LCP class had significantly higher rates of general practitioner treatment, cardiovascular risk based on high-sensitivity C-reactive protein levels, smoking, and dental problems (column C).

The second main finding in Table 4 is that the adolescent-onset class also experienced adult physical health problems. The adolescent-onset class differed significantly from the low class on 7 of 14 physical health outcomes (column D), including hospitalization, lung function, symptoms of chronic bronchitis, smoking (use and dependency), decayed tooth surfaces, and serious injury.

Third, the childhood-limited class was doing relatively well. Only the following 3 differences between the childhood-limited and low classes reached statistical significance: lung function, smoking, and the number of untreated decayed tooth surfaces (column E).

To summarize the results in Table 4, we created a summary variable that indexed the number of physical health problems. The mean number of physical health problems and corresponding effect sizes by trajectory class are displayed at the bottom of Table 4. Moderate to large effect sizes were found when the LCP class was compared with all other classes (d=1.38 vs low; d=0.79 vs childhood limited; and d=0.64 vs adolescent onset). Moderate effect sizes were found between the adolescent-onset and low classes (d=0.64) and between the childhood-limited and low classes (d=0.42).

COMMENT

These findings take us beyond what we know about conduct problems in 4 ways. First, our results provide support for the epidemiological validity of the distinction made in *DSM-IV* regarding childhood- vs adolescent-onset CD. The final GGMM model contained the expected classes

Table 3. Prevalence Rates of Mental Health Problems at 32 Years of Age by Trajectory-Class Membership Among Males in the Dunedin Multidisciplinary Health and Development Study

Mental Health Disorders (n)	Trajectory Class, % of Subjects				Class Comparison, OR (95% CI)				
	Low	CL	AO	LCP	A. LCP vs Low	B. LCP vs CL	C. LCP vs AO	D. AO vs Low	E. CL vs Low
Psychiatric disorders at age 32 y									
Anxiety (490)	10.2	24.8	17.3	32.7	4.3 (2.0-8.9)	1.5 (0.7-3.1)	2.3 (1.1-5.1)	1.9 (0.9-3.6)	2.9 (1.6-5.3)
Major depressive (490)	9.7	10.3	11.2	28.6	3.7 (1.7-7.9)	3.5 (1.5-8.3)	3.2 (1.3-7.6)	1.2 (0.5-2.5)	1.1 (0.5-2.2)
Cannabis dependent (490)	4.9	5.1	15.3	20.4	5.0 (2.0-12.6)	4.7 (1.6-13.9)	1.4 (0.6-3.4)	3.5 (1.6-8.0)	1.1 (0.4-2.9)
Other drug dependent (490)	1.3	0.9	6.1	22.4	21.5 (5.7-80.7)	33.6 (4.2-268.7)	4.4 (1.5-12.9)	4.8 (1.2-19.8)	0.6 (0.07-6.2)
Alcohol dependent (488)	8.0	12.0	19.4	20.8	3.0 (1.3-7.1)	1.9 (0.8-4.7)	1.1 (0.5-2.6)	2.8 (1.4-5.5)	1.6 (0.7-3.3)
Posttraumatic stress (489)	1.3	0.9	3.1	10.2	8.4 (1.9-36.5)	13.2 (1.5-116.0)	3.6 (0.8-15.7)	2.3 (0.5-11.8)	0.6 (0.07-6.2)
Mental health impairment at age 26-32 y									
Received services (490)	11.1	17.9	19.4	32.7	3.9 (1.9-8.1)	2.2 (1.04-4.7)	2.0 (0.9-4.4)	1.9 (1.0-3.7)	1.8 (0.9-3.3)
Received medication (490)	7.1	9.4	15.3	20.4	3.4 (1.4-8.0)	2.5 (1.0-6.3)	1.4 (0.6-3.4)	2.4 (1.1-5.0)	1.4 (0.6-3.0)
Hospitalized (490)	0.9	3.4	6.1	14.3	18.7 (3.7-93.0)	4.7 (1.3-16.9)	2.6 (0.8-8.1)	7.3 (1.4-36.9)	4.0 (0.7-22.0)
Attempted suicide (490)	0.4	3.4	3.1	10.2	25.6 (2.9-224.2)	3.2 (0.8-12.5)	3.6 (0.8-15.7)	7.1 (0.7-69.2)	8.0 (0.9-72.1)
Homeless/taken in (492)	2.6	3.4	6.1	20.4	9.5 (3.3-27.6)	7.2 (2.1-24.4)	3.9 (1.3-11.6)	2.4 (0.8-7.7)	1.3 (0.4-4.7)
Victimization of others at age 32 y									
Partner abuse (449)	11.4	12.7	23.3	20.9	2.1 (0.9-4.8)	1.8 (0.7-4.6)	0.9 (0.4-2.1)	2.3 (1.2-4.5)	1.1 (0.6-2.3)
Controlling abuse (451)	2.9	7.2	11.5	23.3	10.3 (3.5-30.2)	3.9 (1.4-10.7)	2.3 (0.9-6.1)	4.4 (1.6-12.6)	2.6 (0.9-7.8)
Violence, self-report (493)	2.7	5.8	23.2	30.6	16.1 (5.8-44.4)	7.1 (2.7-18.9)	1.5 (0.7-3.1)	11.0 (4.3-28.2)	2.3 (0.7-6.9)
Violence, official at age 26-32 y (496)	0.4	6.0	10.2	32.7	109.1 (14.0-850.0)	7.6 (2.8-19.9)	4.3 (1.8-10.3)	25.6 (3.2-202.7)	14.5 (1.8-118.9)

Summary	No. of Disorders or Episodes, Mean (SD)				Effect Size*				
	Low	CL	AO	LCP	A. LCP vs Low	B. LCP vs CL	C. LCP vs AO	D. AO vs Low	E. CL vs Low
Psychiatric disorders (487)	0.35 (0.69)	0.54 (0.78)	0.72 (0.91)	1.38 (1.54)	1.14 (Large)	0.79 (Moderate)	0.57 (Moderate)	0.49 (Moderate)	0.26 (Small)
Mental health impairment (490)	0.22 (0.59)	0.38 (0.87)	0.51 (0.94)	1.00 (1.31)	1.01 (Large)	0.61 (Moderate)	0.45 (Moderate)	0.40 (Moderate)	0.22 (Small)
Victimization of others (493)	0.16 (0.46)	0.29 (0.53)	0.64 (0.90)	0.94 (1.21)	1.18 (Large)	0.82 (Large)	0.29 (Small)	0.77 (Moderate)	0.27 (Small)

Abbreviations: AO, adolescent onset; CI, confidence interval; CL, childhood limited; LCP, life course persistent; OR, odds ratio.
*Effect sizes were considered small at 0.35 or less, moderate at greater than 0.35 to 0.80, and large at greater than 0.80.

(LCP, adolescent onset, and low), and prevalence rates of the LCP (10.5%) and adolescent-onset classes (19.6%) were as predicted. Finally, the LCP class was, indeed, differentially characterized in childhood by social deprivation, familial liability, and neurodevelopmental deficits.

Second, as anticipated by Robins,[1] conduct problems predicted a variety of negative life outcomes, with this study being the first, to our knowledge, to demonstrate a prospective link between the LCP trajectory and adult health burden. Previous research has focused primarily on the relationship between early-onset CD and future offending behavior, whereas our findings extend the range of outcomes to include mental and physical health status as well as victimization of others. These findings are also consistent with emerging work demonstrating an overlap between predictors of antisocial behavior and predictors of accidents, disease, and mortality.[57-59] What these findings add, however, is the ability to connect a specific developmental pattern of conduct problems to future health problems. For example, the LCP class constituted only 10.5% of the birth cohort males, but they were responsible for 17.5% of the cohort's traffic injuries, 29.4% of the days spent in psychiatric hospitals, 72.2% of the months spent in jail, and 42.3% of the total months where study members were homeless or taken in by friends or relatives.

Our third finding was that individuals who began their involvement in conduct problems in adolescence were at elevated risk for problems in adulthood. This article focused on the adolescent-onset subtype to test the *DSM-IV* construct. However, the taxonomic theory refers to this group as *adolescent limited*, a name that implies desistance from criminal offending behavior during young adulthood. The Figure shows that this group did not self-report fewer CD symptoms than did the LCP class at 26 years of age. However, Table 3 shows that, at 32 years of age, the adolescent-onset group scored below the LCP group on age-relevant measures of offending behavior (eg, court convictions for violence). Thus, the data herein provide mixed evidence regarding whether the adolescent-onset group desists from crime, and this remains an empirical question. The taxonomy anticipated that some individuals on the adolescent-limited pathway would offend longer than others if they attracted snares, ie, factors such as addiction that foster of-

Table 4. Prevalence Rates of Physical Health Problems at 32 Years of Age by Trajectory-Class Membership Among Males in the Dunedin Multidisciplinary Health and Development Study

	Trajectory Class, % of Subjects				Class Comparison OR (95% CI)*				
Physical Health (n)	Low	CL	AO	LCP	A. LCP vs Low	B. LCP vs CL	C. LCP vs AO	D. AO vs Low	E. CL vs Low
Self-report of good/excellent health (494)	57.3	58.3	49.0	36.7	0.4 (0.2-0.8)	0.4 (0.2-0.8)	0.6 (0.3-1.2)	0.7 (0.4-1.2)	1.0 (0.7-1.6)
No. of GP visits, Mean (SD) (493)	1.4 (2.2)	1.8 (2.9)	1.9 (3.5)	3.0 (4.7)	.001	.01	.02	.26	.30
Hospitalized (495)	6.2	10.0	14.1	18.4	3.4 (1.4-8.4)	2.0 (0.8-5.2)	1.4 (0.5-3.4)	2.5 (1.1-5.5)	1.7 (0.8-3.8)
CVD risk (456)	15.7	22.0	15.7	22.9	1.6 (0.7-3.4)	1.1 (0.5-2.4)	1.6 (0.7-3.8)	1.0 (0.5-2.0)	1.5 (0.8-2.7)
C-reactive protein level (455)	11.5	14.7	11.2	27.1	2.9 (1.3-6.2)	2.2 (0.9-4.9)	2.9 (1.2-7.3)	1.0 (0.4-2.1)	1.3 (0.7-2.6)
Lung function, FEV_1/VC, Mean (SD) (482)	77.7 (6.4)	76.1 (7.10)	75.4 (7.3)	76.2 (6.9)	.16	.94	.52	<.01	.04
Chronic bronchitis symptoms (494)	18.1	24.2	29.3	40.8	3.1 (1.6-6.0)	2.2 (1.1-4.4)	1.7 (0.8-3.4)	1.9 (1.1-3.2)	1.4 (0.8-2.5)
Smoker (495)	18.5	35.0	49.5	69.4	10.0 (5.0-20.0)	4.2 (2.1-8.6)	2.3 (1.1-4.8)	4.3 (2.6-7.2)	2.4 (1.4-3.9)
Nicotine dependent (494)	9.3	15.0	31.6	46.9	8.7 (4.2-17.8)	5.0 (2.4-10.6)	1.9 (0.9-3.9)	4.5 (2.4-8.4)	1.7 (0.9-3.4)
Herpesvirus 2 (452)	12.1	17.6	12.4	22.9	2.2 (1.0-4.8)	1.4 (0.6-3.2)	2.1 (0.8-5.3)	1.0 (0.5-2.2)	1.6 (0.8-3.0)
Decayed tooth surfaces, Mean (SD) No. (476)	1.8 (4.2)	3.0 (5.1)	3.8 (5.6)	6.1 (7.1)	<.001	<.001	<.01	.001	.03
Gum disease (476)	17.4	22.8	25.0	42.6	3.5 (1.8-6.9)	2.5 (1.2-5.2)	2.2 (1.1-4.7)	1.6 (0.9-2.8)	1.4 (0.8-2.5)
Serious injury (491)	55.9	57.3	69.4	71.4	2.0 (1.01-3.9)	1.9 (0.9-3.8)	1.1 (0.5-2.3)	1.8 (1.1-3.0)	1.1 (0.7-1.7)
Non–sports-related injury (295)†	68.3	59.7	73.1	88.6	3.6 (1.2-10.9)	5.2 (1.7-16.5)	2.8 (0.9-9.2)	1.3 (0.7-2.4)	0.7 (0.4-1.3)

	No. of Problems, Mean (SD)				Effect Size‡				
Summary	Low	CL	AO	LCP	A. LCP vs Low	B. LCP vs CL	C. LCP vs AO	D. AO vs Low	E. CL vs Low
Physical health problems (441)	3.27 (1.52)	3.96 (1.91)	4.30 (1.81)	5.49 (1.95)	1.38 (Large)	0.79 (Moderate)	0.64 (Moderate)	0.64 (Moderate)	0.42 (Moderate)

Abbreviations: AO, adolescent onset; CI, confidence interval; CL, childhood limited; CVD, cardiovascular disease; FEV_1, forced expiratory volume in 1 second; GP, general practitioner; LCP, life course persistent; OR, odds ratio; VC, vital capacity.

*Data are given as *P* values for number of GP visits, lung function, and decayed tooth surfaces.

†Calculated as a percentage of those with serious injury.

‡Effect sizes were considered small at 0.35 or less, moderate at greater than 0.35 to 0.80, and large at greater than 0.80.

fending behavior. The adolescent-onset group had high rates of substance dependence at 32 years of age, and elsewhere we have shown that substance dependence in this cohort promotes persistence of offending behavior.[60]

Although the prevalence rates of mental and physical health problems are consistently higher among the LCP class, the adolescent-onset class is at risk compared with the low class. In this sense, conduct problems constitute a risk for experiencing adult health problems regardless of age of onset. The adolescent-onset class was also responsible for somewhat more than their share of the overall health burden: they were 19.6% of the cohort, but accounted for 35.0% of the traffic injuries, 36.4% of the days spent in psychiatric hospitals, 25.3% of the months spent in jail, and 28.2% of total months where study members were homeless or taken in by friends or relatives. Although the health of the adolescent-onset class is not as compromised as that of the LCP class, the greater size of the adolescent-onset class accounts for an appreciable fraction of health burden, warranting intensive prevention efforts.

The fourth finding involves the childhood-limited class identified by GGMM. This class included 24.3% of male study members, suggesting that short-term CD symptoms are very common among young boys. Although the childhood-limited class could not be distinguished from the LCP class on CD symptoms at 7 years of age (the confidence intervals overlapped), they were lower on 6 of 7 childhood risk factors (with statistically significant differences on 3 of 7). However, without evidence of persistence, such childhood conduct problems are not sufficient to signal poor outcomes in adulthood.[17,61]

This study has clear limitations. First, it was challenging to construct a developmentally appropriate measure of conduct problems that could be applied from childhood into adulthood. Although we tested and confirmed measurement invariance across our age range, the trajectory shapes were estimated from composite scores and should be interpreted with room for age-specific measurement error. Second, individuals were assigned to classes on the basis of posterior probabilities of their most likely class membership; however, observed pathways often fall between 2 or more estimated trajectories. Individuals do not follow lockstep with the predicted trajectory for their class.[11] These limitations reaffirm the need to view the trajectories as approximations, not precise maps, of the developmental course of conduct problems.

Third, we did not include female study members. The study of the developmental course of girls' conduct problems is currently stalled at the question of whether the taxonomy applies to girls.[17,62-64] Moreover, our initial tests of invariance revealed the complication that symptoms of CD do not index the same construct across ages for girls, suggesting that the sexes could not be fairly compared with each other. A separate set of female-specific analyses is required and will be included in our forthcoming work.

Fourth, this study is based on a single New Zealand cohort. Although the prevalence rates of health and antisocial problems are similar to those in other western nations, and although previous findings about them from this cohort have been replicated,[17] these findings require replication in other countries. As such, generalization of our findings across sex and cultures is an empirical question left for future research.

Finally, our cohort is young in terms of the age-based and cumulative risk for physical health problems. Therefore, we are likely to have underestimated the eventual health burden for the LCP class. Older cohorts and the continued tracking of our cohort can estimate the full extent of the health burden.

With these limitations in mind, certain implications for clinical practice and prevention can be noted. With respect to clinical nosology, our results provide support for the distinction between childhood- and adolescent-onset CD in the *DSM-IV*. The LCP class demonstrated a worse childhood-risk profile and more pervasive health problems in adulthood. The adolescent-onset class, however, was also at risk for health problems, suggesting that the current classification system is well positioned to identify children and adolescents who need treatment to prevent poor adult health.

Our results also support the emphasis on persistence in the *DSM-IV* criteria for CD. Boys on the LCP pathway, characterized by conduct problems that persisted across years, demonstrated poor health outcomes, but their childhood-limited counterparts did not. Based on these findings, it is not clear that the threshold for persistence specified in the *DSM-IV* is long enough to avoid false-positive diagnoses. Instead, the definition of persistence may need to be lengthened from months to years to capture a more developmentally informative window that can differentiate children who are on an LCP developmental trajectory from those on a childhood-limited one.

Our findings also have implications for prevention. First, the costs of CD may be larger than originally thought. These findings provide one of the first comprehensive assessments of the long-term health burden following conduct problems in childhood and adolescence and suggest that the cost estimates of CD may need to be recalibrated to account for many kinds of health care expenses. Second, the presence of early-onset conduct problems alone may not be sufficient grounds to target a child for intervention; boys with childhood-limited conduct problems showed little risk for future health problems. Rather, interventions may be of highest priority for children with conduct problems plus family risk factors or for children whose conduct problems persist for more than 1 year. Finally, adolescence emerges as a second window of opportunity to reduce adult health burden. Taken together, these findings provide yet another reason to make CD a primary target for prevention.[65,66]

Submitted for Publication: February 14, 2006; final revision received June 8, 2006; accepted June 8, 2006.
Correspondence: Terrie E. Moffitt, PhD, Social, Genetic, and Developmental Psychiatry Centre, Institute of Psychiatry, Internal PO Box p080, De Crespigny Park, London SE5 8AF, England (t.moffitt@iop.kcl.ac.uk).
Author Contributions: Dr Odgers takes responsibility for the integrity of the data and the accuracy of the data analysis. All authors had full access to the data.
Financial Disclosure: None reported.
Funding/Support: This study was supported by grants from the US National Institute of Mental Health, the United Kingdom Medical Research Council, the United Kingdom Economic and Social Research Council, the William T. Grant Foundation, the Health Research Council of New Zealand, the Social Sciences and Humanities Research Council of Canada, the Michael Smith Foundation for Health Research, the US National Institute for Dental and Craniofacial Research, and Royal Society–Wolfson Research Merit Awards (Drs Caspi and Moffitt).
Acknowledgment: We thank the Dunedin Study members, the unit research staff, Alan Taylor, MSc, Louise Arsenault, PhD, Daniel Nagin, PhD, and study founder Phil Silva, PhD.

REFERENCES

1. Robins LN. *Deviant Children Grown Up*. Baltimore, Md: Williams & Wilkins; 1966.
2. Caspi A. The child is father of the man: personality continuities from childhood to adulthood. *J Pers Soc Psychol*. 2000;78:158-172.
3. Farrington DP. The development of offending and antisocial behaviour from childhood: key findings from the Cambridge Study in delinquent development. *J Child Psychol Psychiatry*. 1995;36:929-964.
4. Lipsey MW, Derzon JH, eds. *Predictors of Violent or Serious Delinquency in Adolescence and Early Adulthood: A Synthesis of Longitudinal Research*. Thousand Oaks, Calif: Sage Publications; 1998.
5. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*. Washington, DC: American Psychiatric Association; 1994.
6. Moffitt TE. Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychol Rev*. 1993;100:674-701.
7. Moffitt TE. Life-course-persistent and adolescent-limited antisocial behavior. In: Cicchetti D, Cohen DJ, eds. *Developmental Psychopathology: Risk, Disorder, and Adaptation*. Vol 3. 2nd ed. New York, NY: John Wiley & Sons Inc; 2006:570-589.
8. Lahey BB, Waldman ID, McBurnett K. The development of antisocial behavior: an integrative causal model. *J Child Psychol Psychiatry*. 1999;40:669-682.
9. Scott ES, Grisso T. The evolution of adolescence: a developmental perspective on juvenile justice reform. *J Crim Law Criminol*. 1997;88:137-189.
10. Schaffer D. Externalizing disorders workgroup presentation. Presented at the *DSM-V* and *ICD-11* Conference held by the American Psychiatric Institute for Research and Education; February 19, 2004; Bethesda, Md.
11. Nagin DS. *Group-Based Modeling of Development*. Cambridge, Mass: Harvard University Press; 2005.
12. Muthén B, Shedden K. Finite mixture modeling with mixture outcomes using the EM algorithm. *Biometrics*. 1999;55:463-469.
13. Muthén B, Muthén LK. Integrating person-centered and variable-centered analyses: growth mixture modeling with latent trajectory classes. *Alcohol Clin Exp Res*. 2000;24:882-891.
14. Muthén B. Latent variable analysis: growth mixture modeling and related techniques for longitudinal data. In: Kaplan D, ed. *Handbook of Quantitative Methodology for the Social Sciences*. Thousand Oaks, Calif: Sage Publications; 2004:345-368.
15. Nagin DS, Farrington DP, Moffitt TE. Life-course trajectories of different types of offenders. *Criminology*. 1995;33:111-139.
16. Moffitt TE. Life-course-persistent and adolescence-limited antisocial behavior: a 10-year research review and a research agenda. In: Lahey BB, Moffitt TE, Caspi A, eds. *Causes of Conduct Disorder and Juvenile Delinquency*. New York, NY: Guilford Publications; 2003:49-75.
17. Moffitt TE, Caspi A, Rutter M, Silva PA. *Sex Differences in Antisocial Behaviour: Conduct Disorder, Delinquency, and Violence in the Dunedin Longitudinal Study*. New York, NY: Cambridge University Press; 2001.

18. Elley WB, Irving JC. Revised socio-economic index for New Zealand. *New Zeal J Edu Stud*. 1976;11:25-36.
19. Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, Taylor A, Poulton R. Role of genotype in the cycle of violence in maltreated children. *Science*. 2002; 297:851-854.
20. Thurstone TG, Thurstone LL. *The SRA Verbal Form*. Chicago, Ill: Science Research Associates; 1973.
21. Wechsler D. *Manual for the Wechsler Intelligence Scale for Children–Revised*. New York, NY: Psychological Corp; 1974.
22. Moffitt TE, Caspi A, Harkness AR, Silva PA. The natural history of change in intellectual performance: who changes? how much? is it meaningful? *J Child Psychol Psychiatry*. 1993;34:455-506.
23. Caspi A, Silva PA. Temperamental qualities at age three predict personality traits in young adulthood: longitudinal evidence from a birth cohort. *Child Dev*. 1995; 66:486-498.
24. Asendorpf JB, Borkenau P, Ostendorf F, Van Aken MAG. Carving personality description at its joints: confirmation of three replicable personality prototypes for both children and adults. *Eur J Pers*. 2001;15:169-198.
25. Hart D, Atkins R, Fegley S. Personality and development in childhood: a person-centered approach. *Monogr Soc Res Child Dev*. 2003;68:i-vii, 1-109.
26. Robins RW, John OP, Caspi A, Moffitt TE, Silva PA. Resilient, overcontrolled, and undercontrolled boys: three replicable personality types. *J Pers Soc Psychol*. 1996;70:157-171.
27. Costello A, Edelbrock C, Kalas R, Kessler M, Klaric SA. *Diagnostic Interview Schedule for Children (DISC)*. Rockville, Md: National Institute of Mental Health; 1982.
28. *Diagnostic and Statistic Manual of Mental Disorders, Third Edition*. Washington, DC: American Psychiatric Association; 1980.
29. Robins LN, Cottler L, Bucholz KK, Compton W. *Diagnostic Interview Schedule for DSM-IV*. St Louis, Mo: Washington University School of Medicine; 1995.
30. Newman DL, Moffitt TE, Caspi A, Magdol L, Silva PA, Stanton W. Psychiatric disorder in a birth cohort of young adults: prevalence, comorbidity, clinical significance, and new cases incidence from age 11 to 21. *J Consult Clin Psychol*. 1996; 64:552-562.
31. Kessler RC, McGonagle KA, Swartz M, Blazer DG, Nelson CB. Sex and depression in the National Comorbidity Survey, I: lifetime prevalence, chronicity and recurrence. *J Affect Disord*. 1993;29:85-96.
32. Belli RF, Shay WL, Stafford FP. Event history calendars and question list surveys: a direct comparison of interviewing methods. *Public Opin Q*. 2001;65:45-74.
33. Caspi A, Moffitt TE, Thornton A, Freedman D, Amell JW, Harrington H, Smeijers J, Silva PA. The life history calendar: a research and clinical assessment method for collecting retrospective event-history data. *Int J Meth Psych Res*. 1996;6:101-114.
34. Moffitt TE, Caspi A, Krueger RF, Magdol L, Margolin G, Silva PA, Sydney R. Do partners agree about abuse in their relationship? a psychometric evaluation of interpartner agreement. *Psychol Assess*. 1997;9:47-56.
35. Elliott DS, Huizinga D, Menard S. *Multiple Problem Youth: Delinquency, Substance Use, and Mental Health Problems*. New York, NY: Springer-Verlag NY Inc; 1989.
36. Moffitt TE, Caspi A, Harrington H, Milne BJ. Males on the life-course-persistent and adolescence-limited antisocial pathways: follow-up at age 26 years. *Dev Psychopathol*. 2002;14:179-207.
37. Idler EL, Benyamini Y. Self-rated health and mortality: a review of twenty-seven community studies. *J Health Soc Behav*. 1997;38:21-37.
38. Grundy SM, Pasternak R, Greenland P, Smith S, Fuster V. Assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. *J Am Coll Cardiol*. 1999;34:1348-1359.
39. Muñoz A, Gange SJ. Methodological issues for biomarkers and intermediate outcomes in cohort studies. *Epidemiol Rev*. 1998;20:29-42.
40. Caspi A, Harrington H, Milne B, Moffitt TE, Poulton R. Socially isolated children 20 years later: risk for cardiovascular disease. *Arch Pediatr Adolesc Med*. 2006; 160:805-811.
41. Ridker PM, Wilson PWF, Grundy SM. Should C-reactive protein be added to metabolic syndrome and to assessment of global cardiovascular risk? *Circulation*. 2004;109:2818-2825.
42. Pearson TA, Mensah GA, Hong Y, Smith SC Jr; CDC; AHA. CDC/AHA workshop on markers of inflammation and cardiovascular disease: application to clinical and public health practice: overview. *Circulation*. 2004;110:e543-e544.
43. Taylor DR, Fergusson DM, Milne BJ, Horwood LJ, Moffitt TE, Sears MR, Poulton R. A longitudinal study of the effects of tobacco and cannabis exposure on lung function in young adults. *Addiction*. 2002;97:1055-1061.
44. Rasmussen F, Taylor DR, Flannery EM, Cowan JO, Greene JM, Herbison GP, Sears MR. Risk factors for airway remodeling in asthma manifested by a low postbronchodilator FEV1/vital capacity ratio: a longitudinal population study from childhood to adulthood. *Am J Respir Crit Care Med*. 2002;165:1480-1488.
45. Sears MR, Greene JM, Willan AR, Wiecek EM, Taylor DR, Flannery EM, Cowan JO, Herbison GP, Silva PA, Poulton R. A longitudinal, population-based, cohort study of childhood asthma followed to adulthood. *N Engl J Med*. 2003;349:1414-1422.
46. Eberhart-Phillips JE, Dickson NP, Paul C, Herbison GP, Taylor J, Cunningham AL. Rising incidence and prevalence of herpes simplex type 2 infection in a cohort of 26 year old New Zealanders. *Sex Transm Infect*. 2001;77:353-357.
47. Ho DWT, Field PR, Irving WL, Packham DR, Cunningham AL. Detection of immunoglobulin-M antibodies to glycoprotein G-2 by western-blot (Immunoblot) for diagnosis of initial herpes-simplex virus type-2 genital infections. *J Clin Microbiol*. 1993;31:3157-3164.
48. Poulton R, Caspi A, Milne BJ, Thomson WM, Taylor A, Sears MR, Moffitt TE. Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. *Lancet*. 2002;360:1640-1645.
49. McArdle JJ, Epstein D. Latent growth curves within developmental structural equation models. *Child Dev*. 1987;58:110-133.
50. McArdle JJ, Nesselroade JR, Schinka JA, Velicer WF. *Growth Curve Analysis in Contemporary Psychological Research: Handbook of Psychology: Research Methods in Psychology*. Vol 2. New York, NY: John Wiley & Sons Inc; 2003:447-480.
51. Muthén LK, Muthén BO. *Mplus Users Guide*. 3rd ed. Los Angeles, Calif: Muthén & Muthén; 2002.
52. *Mplus, Version 3.13* [computer program]. Los Angeles, Calif: Muthén & Muthén; 2002.
53. Schwartz G. Estimating the dimension of a model. *Ann Statist*. 1978;6:461-464.
54. Akaike H. New look at statistical-model identification. *IEEE Trans Automatic Control*. 1974;19:716-723.
55. Lo YT, Mendell NR, Rubin DB. Testing the number of components in a normal mixture. *Biometrika*. 2001;88:767-778.
56. Arbucle JL. Full information estimation in presence of incomplete data. In: Marcoulides GA, Schumacker RE, eds. *Advanced Structural Equation Modeling, Issues and Techniques*. Hillsdale, NJ: Lawrence A Erlbaum Associates; 1996:243-277.
57. Farrington DP. Crime and physical health: illnesses, injuries, accidents and offending in the Cambridge Study. *Crim Behav Ment Health*. 1995;5:261-278.
58. Shepherd J, Farrington D, Potts J. Impact of antisocial lifestyle on health [published correction appears in *J Public Health (Oxf)*. 2005;27:312-313]. *J Public Health (Oxf)*. 2004;26:347-352.
59. Piquero AR, Gibson CL, Daigle L, Piquero N, Tibbetts SG. Are life-course-persistent offenders at risk for adverse health outcomes? *J Res Crime Delinq*. In press.
60. Hussong AM, Curran PJ, Moffitt TE, Caspi A, Carrig MM. Substance abuse hinders desistance in young adults' antisocial behavior. *Dev Psychopathol*. 2004; 16:1029-1046.
61. Tremblay RE. The development of aggressive behavior during childhood: what have we learned in the past century? *Int J Behav Dev*. 2000;24:129-141.
62. Broidy LM, Nagin DS, Tremblay RE, Bates JE, Brame B, Dodge KA, Fergusson D, Horwood JL, Loeber R, Laird R, Lynam DR, Moffitt TE, Pettit GS, Vitaro F. Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: a six-site, cross-national study. *Dev Psychol*. 2003;39:222-245.
63. Silverthorn P, Frick PJ. Developmental pathways to antisocial behavior: the delayed-onset pathway in girls. *Dev Psychopathol*. 1999;11:101-126.
64. Serbin LA, Cooperman JM, Peters PL, Lehoux PM, Stack DM, Schwartzman AE. Intergenerational transfer of psychosocial risk in women with childhood histories of aggression, withdrawal, or aggression and withdrawal. *Dev Psychol*. 1998; 34:1246-1262.
65. Krug EG, Dahlberg LL, Mercy JA, eds. *World Report on Violence and Health*. Geneva, Switzerland: World Health Organization; 2002.
66. Dodge KA. The science of youth violence prevention: progressing from developmental epidemiology to efficacy to effectiveness to public policy. *Am J Prev Med*. 2001;20(suppl):63-70.