

Predicting Prognosis for the Conduct-Problem Boy: Can Family History Help?

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ABSTRACT

Objective: Many children with conduct disorder develop life-course persistent antisocial behavior; however, other children exhibit childhood-limited or adolescence-limited conduct disorder symptoms and escape poor adult outcomes. Prospective prediction of long-term prognosis in pediatric and adolescent clinical settings is difficult. Improved prognosis prediction would support wise allocation of limited treatment resources. The purpose of this article is to evaluate whether family history of psychiatric disorder can statically predict long-term prognosis among conduct-problem children. **Method:** Participants were male members of the Dunedin Study, a birth cohort of 1,037 children (52% male). Conduct-problem subtypes were defined using prospective assessments between ages 7 and 26 years. Family history interviews assessed mental disorders for three generations: the participants' grandparents, parents, and siblings. **Results:** Family history of externalizing disorders distinguished life-course persistent antisocial males from other conduct-problem children and added significant incremental validity beyond family and child risk factors. A simple three-item family history screen of maternal-reported alcohol abuse was associated with life-course persistent prognosis in our research setting and should be evaluated in clinical practice. **Conclusions:** Family history of externalizing disorders distinguished between life-course persistent versus childhood-limited and adolescent-onset conduct problems. Brief family history questions may assist clinicians in pediatric settings to refine the diagnosis of conduct disorder and identify children who most need treatment. *J. Am. Acad. Child Adolesc. Psychiatry*, 2007;46(10):1240–1249. **Key Words:** psychiatric family history, conduct-disorder diagnosis, conduct-problem trajectories, long-term prognosis.

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Conduct disorder (CD) in childhood and adolescence foretells a wide range of problems in adulthood, including elevated risk of violence, psychiatric disorders, physical-health problems, and dependence on social welfare systems (Fergusson et al., 2005; Kim-Cohen et al., 2003; Moffitt et al., 2002). Correctly identifying juveniles most in need of intervention is complicated, however, by the fact that most children and adolescents who exhibit symptoms of CD desist from antisocial behaviors before they reach young adulthood, and many achieve relatively good outcomes (Robins, 1978; Rutter et al., 2006).

The challenge facing clinicians and researchers is twofold, depending on the developmental period under consideration. First, when a child presents for assessment, the professional's task is to make a differential diagnosis between childhood-onset CD that will be

only childhood-limited versus childhood-onset CD that will have a life-course persistent (LCP) course and pathological prognosis. The age-of-onset distinction cannot help with this task because all child patients by definition have childhood onset. In the absence of advance knowledge of long-term persistence, diagnosing young children does not reliably identify children most in need of intervention and may result in false-positive predictions of prognosis (Lahey et al., 2005). Researchers have tried to distinguish LCP versus childhood-limited trajectory groups by using childhood risk factors, without success (for a review, see Moffitt, 2006).

Second, when an adolescent presents for assessment, the task is to make a differential diagnosis between adolescent-onset CD and childhood-onset CD that is already persistent and well on the way to a pathological adult prognosis. Although both of these subtypes have been shown to experience problems in adulthood, the LCP group engages in more violence and experiences more severe and pervasive mental health and physical health problems (Odgers et al., 2007; Piquero et al., 2007). Moreover, it has been argued that these two subgroups require separate intervention goals and approaches due to their differential childhood origins and age of onset (Howell and Hawkins, 1998; Scott and Grisso, 1997; Vermeiren, 2003). Thus, there is good reason to differentiate these two subgroups during adolescence. It seems obvious to ascertain age at first symptom to make the subtype diagnosis, but this is easier said than done. A clinician may lack access to information about an adolescent patient's symptom history; credible reporters about the adolescents' childhood behavior may not be available, and, even if reporters are at hand, retrospective reports are famously subject to memory failure (Simon and Vonkorff, 1995). Research has shown that the age of onset of conduct problems is generally recalled as years later than it truly was (telescoping; Henry et al., 1994). Official records of age at first police arrest also lag 2 to 5 years behind true age at first illegal act (Moffitt et al., 2001). Good age-of-onset information is hard to obtain.

Thus, during two developmental periods, childhood and mid-adolescence, conduct-problem symptoms alone are not sufficient to distinguish which conduct-problem individuals are most at risk of poor long-term prognosis. The purpose of this article is to examine whether family psychiatric history can assist clinicians working with children and adolescents to best predict

long-term prognosis. It is established that parental (mainly father's) criminality is linked to offspring antisocial behavior (Farrington et al., 2001) and that family liability to antisocial behavior is at the etiological core of CD. Meta-analysis of behavioral genetic studies has shown that CD is under moderate genetic influence (Rhee and Waldman, 2002). Genetic influence is strong for the particular subtype of CD that has an early age of onset and is pervasive, persistent, and severe (Moffitt, 2005a). Research on how genes may contribute to CD is well under way (Moffitt, 2005b), and genetic testing has been proposed for future classification systems (Charney et al., 2002). Taking a cautious view, however, it is unlikely that genetic markers or testing will be available to assist clinicians in predicting prognosis in the near future. In contrast, family history assessments are routinely used to improve prediction of disease prognosis (Yoon et al., 2003) and are more feasible than genetic tests for marking the familial transmission of CD risk. Family history assessments have the potential to be powerful predictors of CD outcome because they combine information about two causes of CD: familial genetic loading plus parents' environmental influences on their children's conduct.

In this article we test whether family history of psychiatric disorders can statistically predict CD prognosis in a birth cohort of children who have been assessed prospectively on conduct-problem symptoms from childhood into adulthood. Well-validated methods were used for gathering family history information (Weissman et al., 2000). Four conduct-problem subtypes (LCP, childhood limited, adolescent onset, and low) were identified using general growth mixture modeling techniques designed to discriminate unique developmental trajectories (Odgers et al., 2007). The first set of analyses tested whether family psychiatric history could distinguish children on the LCP pathway from their childhood-limited and adolescent-onset counterparts. The second set of analyses addressed questions directly relevant to clinical practice. First, the incremental validity of family psychiatric history was evaluated. Second, the efficacy of a brief three-item family history screen for alcohol abuse in differentiating the conduct-problem subtypes was assessed. The brief family history screen was constructed based on items that could be gathered via maternal reports only and were less likely than the reporting of criminal behavior to be subject to social desirability bias.

METHOD

Participants

Participants were members of the Dunedin Multidisciplinary Health and Development Study, a 1-year birth cohort constituted at 3 years of age when investigators enrolled 91% of consecutive eligible births between April 1972 and March 1973 in Dunedin, New Zealand. Here, we report data from 526 males for whom developmental trajectories could be computed, of whom 499 (95% of living males) had available family history data. Cohort families represent the full range of socioeconomic status (SES) in New Zealand's South Island and are primarily white. Follow-up assessments were conducted with informed consent at ages 5, 7, 9, 11, 13, 15, 18, 21, 26, and 32 years of age, when 96% of the living study members were assessed in 2003–2005.

Family psychiatric history data were collected about each participant's biological parents, grandparents, and siblings older than 10 years. Family psychiatric histories were collected in 2003–2005 when the study members were 30 to 33 years of age. Data on 4,001 family members of the males in the Dunedin study were used (average of 8.0 family members; range 3–16), including 1,931 first-degree relatives (499 biological mothers, 495 biological fathers, and 937 full siblings) and 2,070 second-degree relatives (945 biological grandmothers, 918 biological grandfathers, and 207 half-siblings).

The Otago, Wisconsin, and Maudsley Ethics Committees approved this research, and study members and their parents gave informed consent before participating.

Measures

Developmental Subtypes of Antisocial Conduct Problems. Developmental subtypes of antisocial conduct problems were identified in our previous work using general growth mixture modeling (Odgers et al., 2007). Conduct problems were assessed prospectively at ages 7, 9, 11, 13, 15, 18, 21, and 26. Six key symptoms of CD were scored as being present or absent at each age: physical fighting, bullying others, destroying property, telling lies, committing truancy, and stealing. Four conduct-problem subtypes were identified: an LCP class who initiated conduct problems in childhood and persisted into adulthood, an adolescent-onset class whose conduct problems began during adolescence, a childhood-limited class whose conduct problems started in childhood but subsequently desisted, and a low class whose conduct problems remained low throughout development (Fig. 1).

Family History of Psychiatric Problems. Up to three informants provided reports on each family member (e.g., the participant and both of their parents; 77.6% of male study members had three reporters, 17.8% had two, and 4.6% had one). Family psychiatric history was assessed using the Family History Screen, a valid and reliable measure of psychiatric family health history (Weissman et al., 2000). Use of more than one informant per family to identify cases using this protocol results in median sensitivity across disorders of 68.2 and median specificity of 86.8. The κ values for the protocol's test-retest reliability across a 15-month period range from .52 (for anxiety) to .66 (for drugs); other available κ values were .56 (depression), .53 (CD), and .61 (alcohol; Weissman et al., 2000). To minimize potential underreporting, the Family History Screen uses pairs of questions to ascertain each symptom. First, a broadly sensitive introductory screen question is asked to stimulate memory and give the respondent time to reflect (e.g., "Has anyone

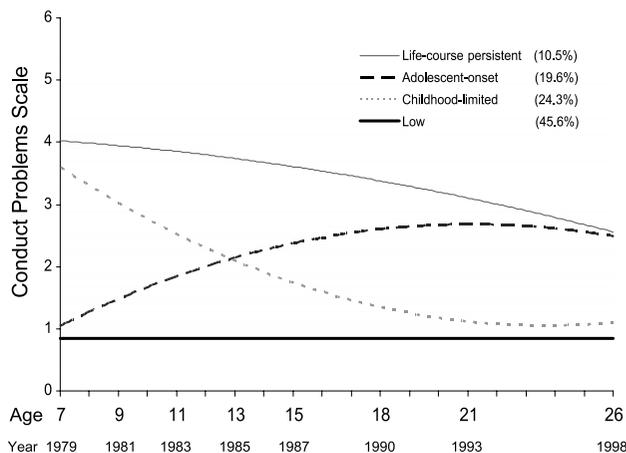


Fig. 1 Conduct-problem subtypes from ages 7 to 26 ($N = 526$ males). A *DSM-IV* diagnosis of conduct disorder requires the presence of three or more conduct-problem symptoms. The symptom counts shown represent a conservative estimate of the average symptom count for each subgroup due to the fact that only six conduct problem symptoms were included in the conduct-problems scale.

on the list of family members ever had a sudden spell or attack in which they felt panicked?"). If any family members are named in response to the introductory question, then it is followed by a second, narrower symptom definition question (e.g., "Has anyone has several attacks of extreme fear or panic, even though there was nothing to be afraid of?"). For data analysis purposes, only the second questions were used.

To broaden the coverage of the Family History Screen, we added items from the Diagnostic Interview Schedule (Robins et al., 1995), the Short Michigan Alcoholism Screening Test (Selzer et al., 1975), and the Drug Abuse Screening Test (Skinner, 1983). A checklist of psychiatric conditions commonly understood by the public (e.g., alcoholism, attention deficit disorder, depression) and an item asking whether family members had ever been a smoker was also added. Here, we report family history for five externalizing spectrum disorders: CD/antisocial personality disorder (CD/ASPD, eight items), alcohol abuse (three items), drug abuse (three items), smoking (one item), and attention-deficit/hyperactivity disorder (ADHD; three items), and two internalizing spectrum disorders: major depression (four items) and anxiety (13 items on generalized anxiety, panic, agoraphobia, phobia, and obsessive-compulsive disorder). A family member was considered to have a positive history of a disorder if one or more of the disorder's items were endorsed by at least two reporters when three reporters were available or by one reporter when fewer than three reporters were available. Each participant's family's liability for disorder was then calculated as the proportion of members in the family with a positive history of disorder. To take account of genetic relatedness, second-degree relatives are considered to be half a family member for the purposes of calculating this proportion. For example, if a family comprises four grandparents, two parents, one full sibling, and one half sibling, of whom one grandparent, one parent, and one full sibling were reported to have alcohol abuse, the proportion of family members with alcohol abuse would be 0.45 (i.e., 2.5, two first-degree relatives plus one second-degree relative, divided by 5.5, three first-degree relatives plus five second-degree relatives). For ease of comparison across disorders, family liability scores for each disorder were z standardized (mean 0 and SD 1).

Family and Child Risk Factors

SES was measured as the highest of father's or mother's occupation using a 6-point scale for New Zealand (Elley and Irving, 1976); 21% of the families were classified as low SES.

Maltreatment was measured using staff observations of rejecting mother-child interaction at age 3, parental reports of harsh discipline at ages 7 and 9, two or more changes in primary caregiver to age 11, and retrospective reports by study members at age 26 of injurious physical abuse or unwanted sexual contact before age 11; 9% of the sample had two or more indicators of maltreatment (Caspi et al., 2002).

Mother's IQ was tested using the Science Research Associates verbal test (Thurstone and Thurstone, 1973) when the children were age 3, standardized to a population mean of 100 (SD 15). Low mother IQ was defined as a score of <85 on the standardized Science Research Associates.

Child IQ was tested using the WISC-R (Wechsler, 1974) at ages 7, 9, 11, and 13, and the four values were averaged to enhance reliability, standardized to a population mean of 100 (SD 15; Moffitt et al., 1993). Low child IQ was defined as a score <85 on the standardized WISC-R.

Undercontrolled temperament was measured through staff ratings after observing the child in a 90-minute testing session with an unfamiliar examiner at age 3. Factor and cluster analyses reduced these ratings to three temperament types, including the undercontrolled type (Caspi and Silva, 1995) since replicated in other samples (Asendorpf et al., 2001; Hart et al., 2003; Robins et al., 1996).

ADHD was measured using the Diagnostic Interview for Children (Costello et al., 1982) at ages 11, 13, and 15. Diagnoses were made according to *DSM-III* (American Psychiatric Association, 1980) and confirmed through parent or teacher report (Moffitt et al., 2001); 9.6% of the males met diagnostic criteria for ADHD.

Data Analysis

Two sets of analyses were performed. The first, ordinary least-squares regression, was applied to test whether individuals on the LCP pathway could be differentiated from their childhood-limited or adolescent-onset counterparts on family history of externalizing or internalizing disorders. Analyses were repeated with siblings excluded from the family liability scores to rule out reverse causation due to potential influence of the study member on his siblings. Analyses were also repeated using only grandparents' family history information to control for the possibility that children's behavior problems influenced their parent's mental health (reverse causation). Next, the specificity of each externalizing (CD/ASPD, alcohol dependence, drug dependence, smoking, ADHD) and internalizing (depression, anxiety) disorder in separating those on the LCP pathway from their childhood-limited or adolescent-onset counterparts was evaluated.

The second set of analyses sought to maximize the translation of findings to clinical settings where it may not be feasible to complete a full family history assessment. Multiple logistic regression analyses were applied to test whether family history of externalizing problems demonstrated incremental validity when considering other family and child risk factors available to clinicians in pediatric and adolescent settings. Next, we tested whether a brief three-item family history screen based on mothers' reports of family members' alcohol abuse functioned in the same way as our more lengthy family history assessment and could thus be used to differentiate

those on the LCP pathway from their childhood-limited and adolescent-onset counterparts. The brief family history screen was derived to mirror three restrictions faced by clinicians working with children in pediatric settings: limited time, a restricted number of informants from whom family history information can be obtained, and, often, an unwillingness on the part of parents to report on involvement in illegal or criminal activities (in contrast to alcohol use, which may be more socially acceptable).

RESULTS

Can Family Psychiatric History Identify Children and Adolescents on the LCP Pathway?

Figure 2a presents family liability scores for externalizing problems by conduct-problem subtypes and illustrates that those on the LCP pathway had significantly higher family liability scores compared to their childhood-limited ($\beta = 0.85$, $p < .001$) and

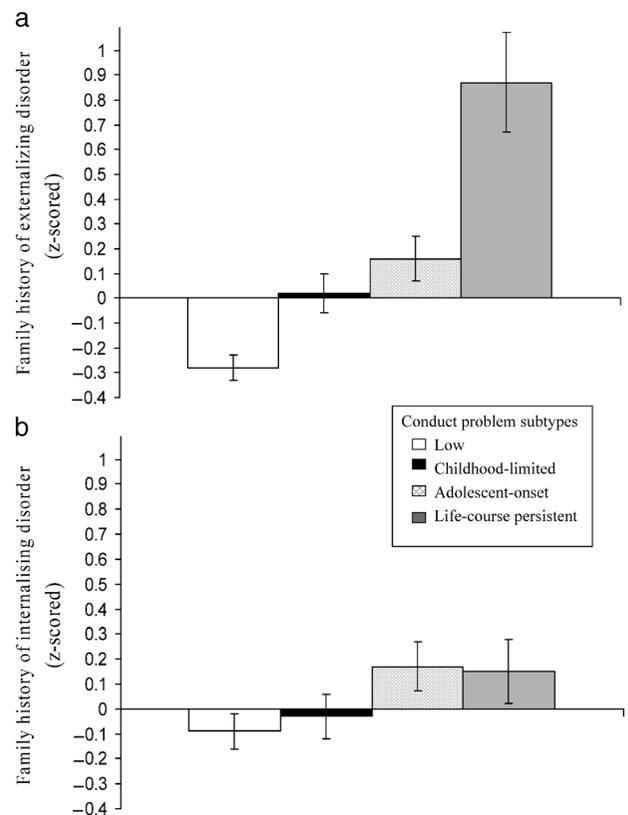


Fig. 2 Family history of externalizing (a) and internalizing (b) disorders by conduct-problem subtypes. Family history of externalizing disorders was computed as the mean of the family liability scores for each of the five externalizing disorders (conduct disorder/antisocial personality disorder, alcohol abuse, drug abuse, smoking, attention-deficit/hyperactivity disorder). Family history of internalizing disorders was computed as the mean of the family liability scores for depression and anxiety. Scores have been z score standardized so that the mean = 0 and SD = 1.

adolescent-onset ($\beta = 0.71, p < .001$) counterparts. Results held after siblings were removed from the family liability scores and when only grandparents' externalizing problems were included in the family liability score.

Figure 2b presents family liability scores for internalizing problems by conduct-problem subtype and illustrates that those on the LCP pathway could not be distinguished from childhood-limited ($\beta = 0.18, p = .28$) or adolescent-onset ($\beta = -0.02, p = .89$) conduct-problem subtypes.

Figure 3a to e presents family liability scores for each type of externalizing disorder by conduct-problem

subtype and highlights three main findings. First, those on the LCP pathway had the highest family liability score for each type of externalizing disorder. Second, the children on an LCP versus childhood-limited pathway could be differentiated on the majority (three of five) of family liability scores for externalizing disorders; the LCP subgroup had a significantly greater proportion of family members with histories of CD/ASPD ($\beta = 0.91, p < .001$), alcohol abuse ($\beta = 0.77, p < .001$), and drug abuse ($\beta = 0.58, p < .001$), with a trend toward a greater proportion of family members with smoking ($\beta = 0.30, p = .07$) and ADHD ($\beta = 0.30, p = .07$). Third, adolescents on the LCP versus adolescent-onset pathway

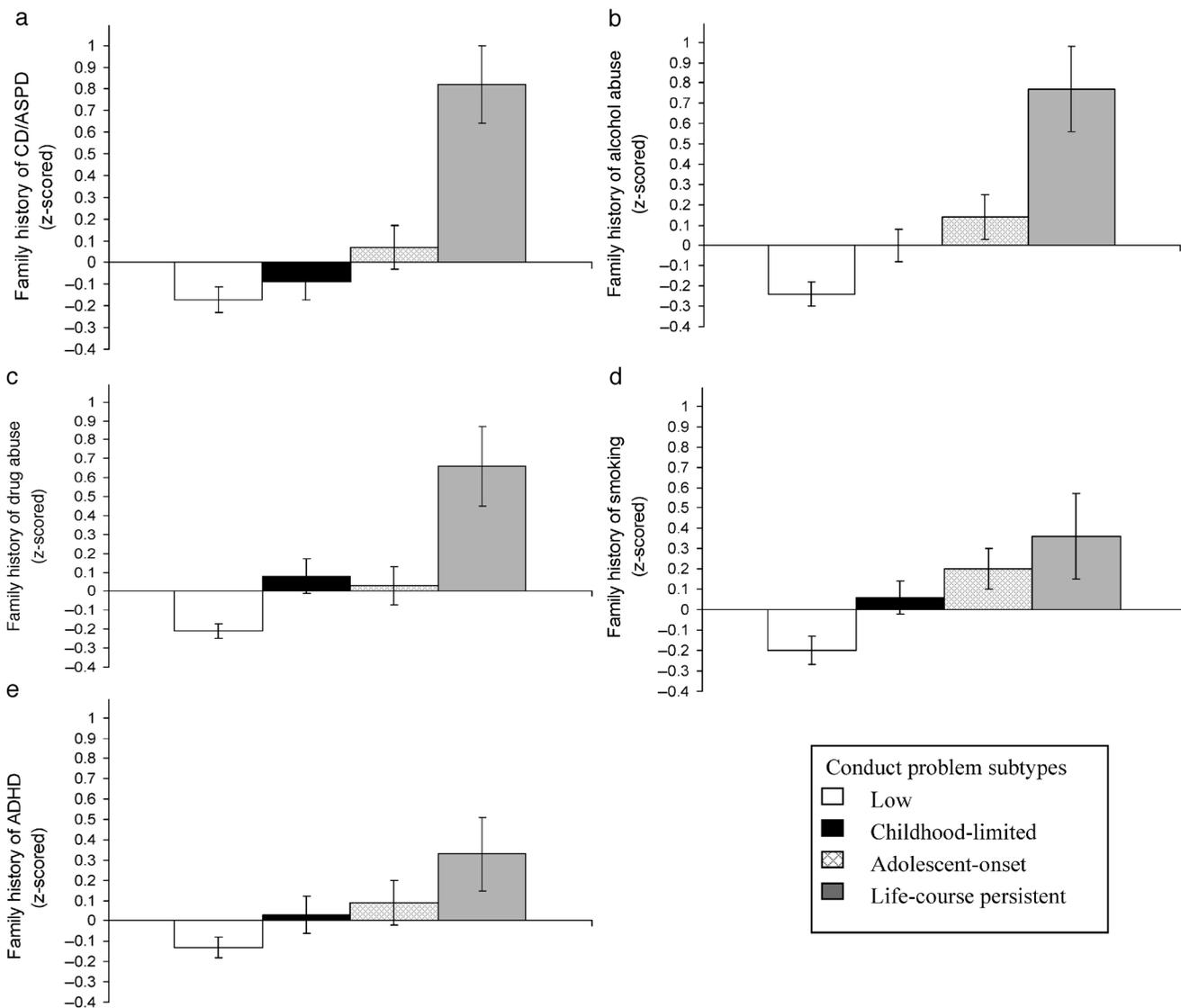


Fig. 3 Family history of specific externalizing disorders by conduct-problem subtypes: (a) conduct disorder/antisocial personality disorder, (b) alcohol abuse, (c) drug abuse, (d) smoking, (e) attention-deficit/hyperactivity disorder. Scores have been z score standardized so that the mean = 0 and SD = 1.

TABLE 1

Does Family History of Externalizing Disorders Have Incremental Validity When Distinguishing LCP Conduct-Problem Subtypes?

	β	SE	<i>p</i>	OR	95% CI	
					Lower	Upper
LCP vs. childhood-limited subtype						
Family history of externalizing disorders	0.64	0.19	<.01	1.90	1.31	2.76
Low SES	0.43	0.47	.37	1.54	0.61	3.88
Maltreatment	0.86	0.52	.10	2.36	0.85	6.59
Mothers IQ (low)	0.20	0.47	.67	1.23	0.49	3.10
Child IQ (low)	-0.18	0.50	.72	0.83	0.31	2.24
Undercontrolled temperament	-0.41	0.53	.44	0.66	0.24	1.87
Child ADHD diagnosis	1.53	0.50	<.01	4.62	1.75	12.19
Constant	-1.62	0.31	.000	0.20		
LCP vs. adolescent-onset subtype						
Family history of externalizing disorders	0.51	0.20	.01	1.66	1.12	2.46
Low SES	0.06	0.50	.90	1.07	0.40	2.84
Maltreatment	1.44	0.62	.02	4.23	1.27	14.15
Mothers IQ (low)	0.65	0.53	.23	1.91	0.67	5.41
Child IQ (low)	-0.27	0.61	.66	0.78	0.23	2.53
Undercontrolled temperament	0.23	0.57	.69	1.25	0.41	3.84
Child ADHD diagnosis	2.53	0.63	<.01	12.56	3.63	43.46
Constant	-1.67	0.33	<.01	0.19		

Note: Statistically significant results ($p < .05$) are shown in bold. Estimates are adjusted to account for all of the other independent variables in the model. LCP = life-course persistent; SES = socioeconomic status; ADHD = attention-deficit/hyperactivity disorder.

could also be differentiated on the majority (three of five) of family liability scores for externalizing disorders; those on the LCP pathway had a significantly greater proportion of family members with histories of CD/ASPD ($\beta = 0.75, p < .001$), alcohol abuse ($\beta = 0.62, p < .001$) and drug abuse ($\beta = 0.63, p < .001$).

Could Family History of Externalizing Disorders Help Clinicians Predict Prognosis for the Conduct-Problem Child?

To address this question, two sets of analyses were performed. First, the incremental validity of family history of externalizing problems was tested controlling for commonly used family and child risk factors (low SES, maltreatment, low maternal IQ, low child IQ, undercontrolled temperament, ADHD). As shown in Table 1, family history of externalizing problems was independently associated with whether male children followed an LCP versus a childhood-limited conduct-problem trajectory. Family history of externalizing problems also statistically predicted whether male adolescents followed an LCP versus an adolescent-

onset conduct-problem trajectory when accounting for the contribution of other family- and child-risk factors.

Second, we tested whether a brief three-item family history screen for alcohol abuse, based on maternal reports about six family members (both biological parents and all four grandparents), could distinguish those on the LCP pathway from their childhood-limited and adolescent-onset counterparts. The family history screen included the following items: "Has ____ ever had any treatment or been in hospital for drinking?" "Has ____ ever had alcoholism?" and "Has ____ ever had a drinking problem or did other people think he/she had a drinking problem?" As illustrated in Figure 4, the brief family history screen functioned in the same way as our comprehensive family history assessment protocol and differentiated individuals on an LCP versus childhood-limited ($\beta = 0.73, p < 0.001$) and adolescent-onset ($\beta = 0.44, p = .01$) conduct-problem trajectory.

In childhood, when the challenge is to separate those on the LCP versus childhood-limited conduct-problem pathway, the brief family history screen had a positive

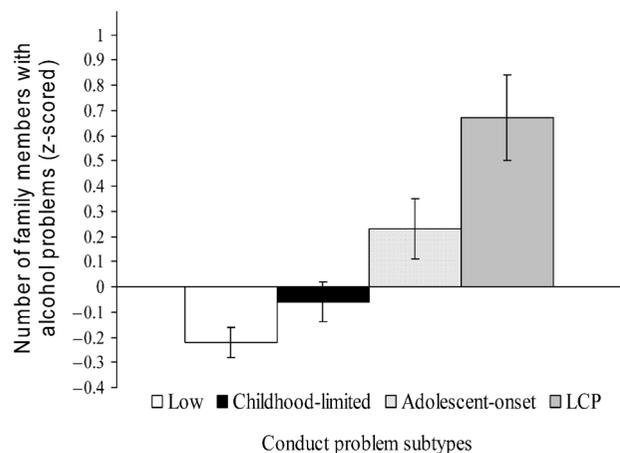


Fig. 4 Can a three-item family history screen identify individuals on the LCP pathway? Scores have been z score standardized so that the mean = 0 and SD = 1. LCP = life-course persistent.

predictive value of 69.2% and a negative predictive value of 73.2%. That is, 69.2% of study members with a positive family history screen were following the LCP pathway, and 73.3% of study members with a negative family history screen were following the childhood-limited (versus LCP) conduct problems. In adolescence, when the challenge is to separate those on the LCP versus adolescent-onset conduct-problem pathway, the brief family history screen had a positive predictive value of 60.0% and a negative predictive value of 69.0%. The brief family history screen had high specificity (>96%) but low sensitivity (<20%). That is, virtually all of the study members with a positive score on the family history screen were on an LCP conduct-problem trajectory (versus those with childhood-limited or adolescent-onset conduct problems), but a significant number of those children on an LCP pathway were not captured using a family history screen alone.

DISCUSSION

Findings from this study advance what is known about predicting prognosis for the conduct-problem child in three ways. First, our results demonstrate that a family history of externalizing disorders, but not internalizing disorders, differentiates male children on an LCP versus childhood-limited conduct-problem trajectory; this association was large and held after excluding siblings and grandparents from the family history liability score. Support was also found for

incremental validity, suggesting that the family psychiatric history of externalizing disorders provides an independent source of information for clinicians trying to predict prognosis for male children. Prior research has failed to identify reliable family and child risk factors that differentiate these two subtypes; as such, family psychiatric history information, if it can be assessed reliably during childhood, may assist clinicians working with young children in pediatric settings.

Second, our results demonstrate that a family history of externalizing disorders, but not internalizing disorders, differentiates adolescent males on an LCP versus adolescent-onset conduct-problem trajectory; again, this association was large and held after excluding siblings and grandparents from the family history liability score. Support was also found for incremental validity, suggesting that family psychiatric history provides an independent source of information for clinicians trying to predict prognosis for male adolescents. Although prospective longitudinal studies have identified a number of family and child risk factors that distinguish these two subtypes (Moffitt, 2006), these factors may be difficult to reliably assess in clinical settings during adolescence. Similarly, although age-of-onset information is a cardinal feature of the *DSM-IV* (American Psychiatric Association, 1994) criteria for distinguishing between child-onset versus adolescent-onset subtypes, age-of-onset information is often unreliable when gathered in adolescence using retrospective recall (Rutter et al., 2006). Thus, family history information may serve as an additional tool for clinicians when predicting prognosis for adolescents in their care, particularly when the adolescent is being seen for the first time (as is common).

Third, our findings demonstrated that a simple three-item family history screen of mothers' reports about alcohol abuse in the child's parents and grandparents was able to statistically predict LCP prognosis. The brief family history screen was created to mirror restrictions faced by clinicians working with children in pediatric settings, such as limited time, a restricted number of informants, and, often, an unwillingness of parents to report on involvement in illegal or criminal activities. In our research setting a brief three-item family history screen functioned in the same way as our full family history assessment protocol in differentiating individuals on an LCP pathway and should be evaluated further in clinical settings.

This study is unique in that it includes a birth cohort of children who have been assessed prospectively on conduct-problem symptoms from childhood into adulthood and well-validated multiple-informant methods for gathering family history information from three reporters about three generations. In addition, conduct-problem subtypes were defined using advanced longitudinal methods to maximize the classification of study members according to the *DSM-IVCD* criteria. To our knowledge, this is the first study to integrate advances in both family history assessment methods and trajectory-based modeling to inform clinical decision making.

Limitations

This study also has clear limitations. First, we did not include females in these analyses. Research on females has demonstrated stronger continuity between conduct problems in childhood and later antisocial behavior, suggesting that distinguishing between childhood-limited versus LCP conduct problems is less of a concern for clinicians working with female children (Costello et al., 2003; Odgers et al., in press; Schaeffer et al., 2006). Nevertheless, this is an empirical issue that should be addressed in future research.

Second, these findings are based on a single New Zealand cohort. The cohort's prevalence rates of health and antisocial problems are similar to those in other Western nations, and previous findings from this cohort have been replicated elsewhere (Moffitt et al., 2001). However, this is a primarily white sample and, as such, the extension of our findings to ethnic minority populations is not known.

Third, it is clear that family history assessments have their drawbacks. Clinicians are unlikely to have the time or resources to carry out detailed family history interviews, reliable informants may not be available, and social desirability, memory failure, and lack of knowledge may lead parents to underreport family history information, particularly with respect to behaviors that carry a social stigma such as criminal behavior and, although to a lesser extent, alcohol abuse, which is not illegal. In particular, it is possible that our family history assessment was biased due to the collection of family history information when the study members were adults versus when they were children. Moreover, these findings are based on an epidemiological birth cohort versus a clinical cohort.

Ideally, future research will assess family psychiatric history during childhood in prospectively followed clinical samples to gauge how well our findings translate from the laboratory to real life (Weisz et al., 1992).

Clinical and Policy Implications

With the above limitations in mind, implications for clinicians working with children and adolescents can be noted. First, family history of externalizing disorders may assist clinicians in predicting prognosis in two developmental periods. In childhood, when conduct problems are common, family history information may help separate children who are at the highest risk of following a persistent and severe conduct-problem trajectory from children whose conduct problems are limited to childhood and who have a more favorable long-term prognosis. Likewise, in adolescence, family history information may help to separate youths on an LCP trajectory from their more transient adolescent-onset peers, who have been shown to obtain better adult outcomes and are likely to require separate intervention approaches (for a review, see Moffitt, 2006). Thus, there is good reason to continue improving our ability to predict prognosis for both subtypes of conduct-problem adolescents.

Second, family history information may guide primary prevention efforts and ensure the wise allocation of resources by identifying families most in need of interventions and support at an early stage, possibly as part of prenatal counseling. Moreover, the most effective interventions for CD invariably require the participation of parents (McCart et al., 2006), but antisocial parents are at highest risk of terminating treatment (Kazdin et al., 1997). Thus, family history assessments may assist treatment efforts by informing clinicians about both the child's and the family's potential amenability and responsiveness to treatment.

Third, family history information may improve the predictive accuracy of population-based screening tools for CD. Attempts to identify children at high risk of CD in the general population have demonstrated that conduct problem symptoms alone are not sufficient (Bennett and Offord, 2001). Our findings suggest that family history information may be an ideal candidate for improving predictive accuracy within both population and clinical settings. However, future research is required to evaluate the combined diagnostic accuracy of family history information when administered

alongside already established assessment and screening tools for CD, such as the Early Assessment Risk List for Boys (Augimeri et al., 2001).

Fourth, family history of externalizing disorders should be evaluated to determine whether it could improve classification in psychiatric diagnostic systems (Moffitt et al., in press; available from t.moffitt@iop.kcl.ac.uk). Family history information has proven utility in medical settings and is routinely used for population-wide prevention of cardiovascular disease and related conditions (Hunt et al., 2003). Like family medical history, family history of externalizing disorders demonstrated incremental validity in our research setting and differentiated a small subset of children who may benefit most from early targeted interventions. In preparation for *DSM-V*, additional research is needed in clinical samples to determine whether family psychiatric history helps to predict long-term prognosis and identify children who need treatment most and, if so, whether family psychiatric history can be assessed reliably by clinicians in treatment settings.

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Masculine Beliefs, Parental Communication, and Male Adolescents' Health Care Use Arik V. Marcell, MD, MPH, Carol A. Ford, MD, Joseph H. Pleck, PhD, Freya L. Sonenstein, PhD

Objectives: Male adolescents frequently become disconnected from health care, especially as they get older, which limits physicians' abilities to address their health needs and results in missed opportunities to connect them to the health care system as they enter adulthood. In this study we tested the ability of modifiable (beliefs about masculinity, parental communication, sex education, and health insurance) and nonmodifiable (age, race/ethnicity, and region of residence) factors to prospectively predict health care use by male adolescents. *Patients and Methods:* We conducted a prospective analysis of data from 1677 male participants aged 15 to 19 years who completed the National Survey of Adolescent Males, a household probability survey conducted throughout the United States in 1988 (wave 1, participation rate: 74%) and in 1990–1991 (wave 2, follow-up rate: 89%). We present percentages and adjusted relative risks of the factors that predict male adolescents' self-report of a physical examination by a regular provider in the past year measured at wave 2. *Results:* On average, 1067 (66%) of 1677 male adolescents at wave 2 reported having a physical examination within the last year. Factors associated with a lower likelihood of a physical examination included living in the South, Midwest, and West; being older in age; and holding more traditional masculine beliefs. Factors associated with a higher likelihood of a physical examination included communicating about reproductive health with both parents and being insured. Male adolescents who were sexually active or engaged in ≥ 2 other risk behaviors had neither a higher nor lower likelihood of a physical examination. *Conclusions:* Efforts to enhance male adolescents' health through health care should include work to modify masculine stereotypes, improve mothers' and fathers' communication about health with their sons, expand health insurance coverage, and identify interventions to connect male adolescents at increased risk for health problems with health care. **Pediatrics** 2007;119:e966–e975.