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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

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 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.

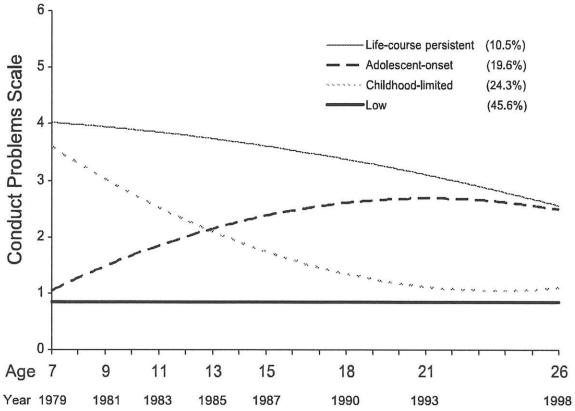

Fig. 1 Conduct-problem subtypes from ages 7 to 26 (N = 526 males). A *DSM-IV* diagnosis of conduct disorder requires the presence of three or more conduct-problem symptoms. The symptom counts shown represent a conservative estimate of the average symptom count for each subgroup due to the fact that only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

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).

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

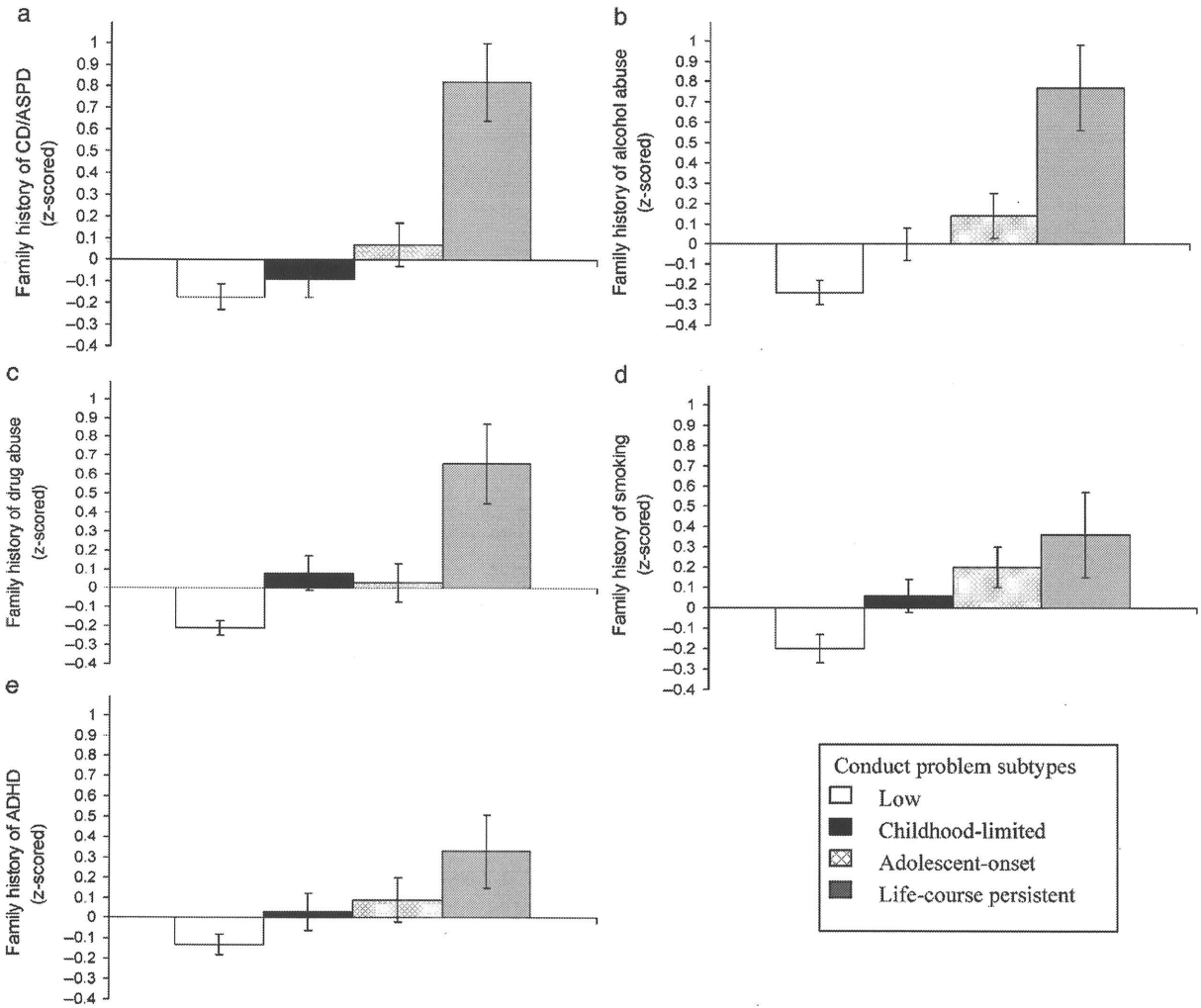

Fig. 3 Family history of specific externalizing disorders by conduct-problem subtypes: (a) conduct disorder/antisocial personality disorder, (b) alcohol abuse, (c) drug abuse, (d) smoking, (e) attention-deficit/hyperactivity disorder. Scores have been *z* score standardized so that the mean = 0 and SD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

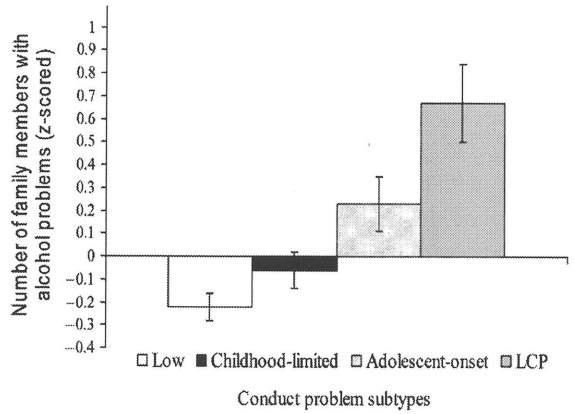

Fig. 4 Can a three-item family history screen identify individuals on the LCP pathway? Scores have been *z* score standardized so that the mean = 0 and SD = 1. LCP = life-course persistent.

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.

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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