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Children With Early-Onset Disruptive Behavior: Parental Mental Disorders Predict Poor Psychosocial Functioning in Adolescence

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Objective: Parental mental disorders (MD) and child early-onset disruptive behavior (DB) are well-established risk factors for poor outcomes in adolescence. However, it is not clear whether parental MD increases risk of future maladjustment among children who already display DB.

Method: Parents of 9-year-old children reported on child DB, whereas a patient registry was used to determine parental MD. At follow-ups at ages 15 (n = 6,319) and 18 (n = 3,068) years, information about various problems were collected via registries, parent-, and self-reports.

Results: In the total sample, child DB was related to all outcomes (mean odds ratio [OR] = 1.18; range = 1.07−1.51; p values < .01), paternal MD to criminality, aggression, truancy, poor school performance, and a cumulative risk index of poor functioning, and maternal MD to peer problems, rule breaking, and truancy (mean OR = 1.67; range = 1.19−2.71; p values < .05). In the subsample of children with DB, paternal MD predicted criminality, consequences of antisocial behavior, truancy, poor school performance, and cumulative risk, whereas maternal MD predicted peer problems (mean OR = 1.94; range = 1.30−2.40; p values < .05).

Conclusion: This study provides novel evidence that parental MD places 9-year-olds with DB at risk for negative outcomes in adolescence. In addition, paternal MD is a better predictor than maternal MD, regardless of child DB at age 9, suggesting that fathers should be given increased attention in future research. Treatment-as-usual of children with DB could be augmented with additional screening and, if necessary, treatment of mental health problems in their parents.

Key words: longitudinal studies, conduct disorder, oppositional defiant disorder, aggression, child of impaired parents

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It is well documented that children with early-onset disruptive behavior (DB), including oppositional defiant and conduct disorder symptoms, have a high risk of adverse psychosocial outcomes in adolescence and adulthood, such as school dropout, criminality, substance abuse, reduced social skills, and mental health problems.[1-3] There are also clear indications that parental mental disorders (MDs) are involved in the onset[4] and maintenance[5,6] of childhood DB and other negative psychosocial outcomes.[7] Children with DB often cause emotional distress and discord in the family,[8] suggesting that childhood DB increases the risk of MD in parents.[9] However, it is not well researched whether parental MD increases the risk of poor psychosocial outcomes in children who already display early-onset DB, mainly because studies did not test interaction effects between child DB and parental MD in their total sample[10] or did not test the prognostic usefulness of parental MD in a subsample of youth with DB.[11] This lack of research is surprising, as parental MDs have been considered to constitute a major risk factor for treatment failure of childhood DB.[12]

We are aware of only one study that has addressed this topic. In a sample of 132 preschoolers aged 3 years with DB, Breaux *et al.* showed that indices of maternal and paternal psychopathology were predictive of parent ratings of child externalizing and internalizing problems and social skill deficits 3 years later.[13] These findings suggest that parental MD in children with DB is a risk factor for poor prognoses. Yet, the Breaux *et al.* study[13] had some notable limitations that must be addressed in future work on this topic. First, parents were the sole informants, and this shared method variance increased the likelihood of revealing significant associations between parental psychopathology

and child functioning. Second, parental psychopathology was assessed by means of dimensional measures, and the findings therefore may not generalize to parents with clinical diagnoses. Third, Breaux *et al.* used a 3-year follow-up interval to study outcomes of preschoolers with parents with an MD. Therefore, it is uncertain whether children with DB who have parents with MDs are at an increased risk for outcomes assessed in adolescence.

Both early-onset DB and parental MDs are risk factors for a variety of problems in adolescence. Therefore, we first tested the hypothesis that DB and parental MDs predict poor psychosocial functioning in the total sample of children. Crucially, our main aim was to investigate whether 9-year-old children with DB are at a greater risk for maladjustment in middle (age 15) and late (age 18) adolescence when considering maternal and paternal MD status. As such, we hypothesized that prospective relations between parental MD and outcomes in a subsample of children with DB would emerge. In line with prior work,[10] we examined whether child DB and parental MD predicted each outcome separately as well as a cumulative index of poor functioning.

METHOD

Participants

The Child and Adolescent Twin Study in Sweden (CATSS) is a nationwide longitudinal study that targets all twins born in Sweden since July 1992.[14] Parents of twins were administered the Autism-Tics, AD/HD and other Comorbidities inventory (A-TAC) by telephone in connection with the twins' ninth birthday (twins born from July 1, 1992 to June 30, 1995 were included at age 12 years). The families were contacted again in connection with the twins' 15th birthday and again at age 18. The follow-up at 15 years includes twins born in 1994 and onward, whereas the follow-up at 18 years includes twins born in 1992 and onward. At both follow-up assessments, at least one parent and both twins were invited to participate.

At baseline (age 9), parents completed the A-TAC as described below (see Measures) for 8,906 twins (born in 1992−1999), of whom 7,105 participated at the first follow-up and 4,492 at the second follow-up. For the purpose of the present investigation, participants were selected for whom outcome measures of interest were available at age 15 (first follow-up), resulting in a sample of 6,319 children, and for whom outcome measures of interest were available at age 18 (second follow-up), resulting in a sample of 3,068 children. A subsequent selection of children with DB resulted in a subsample of 2,215 children at the first follow-up, and a subsample of 1,190 children at the second follow-up. Descriptive information of all samples is provided in Tables 1 and 2.

Baseline Measures at Age 9 Years

Parent-Reported Disruptive Behavior. Parent-reported disruptive behavior (DB) of the child was assessed using the A-TAC, which consists of 96 questions covering common child and adolescent psychiatric disorders, including oppositional defiant disorder (ODD) and conduct disorder (CD).[15] The A-TAC ODD and CD subscales consist of five gate questions, each asking a parent about lifetime presence of ODD and CD symptoms in his/her child, respectively. The answering options are coded as 0 ("no"), 0.5 ("yes, to some extent"), or 1 ("yes"). All A-TAC questions are included in Table S1, available online.

Registered Parental Mental Disorder. The presence of parental mental disorder (MD) was based on information retrieved from the National Patient Register (NPR). The NPR has been registering psychiatric inpatient admissions since 1973 and outpatient consultations since 2001. Mental disorders are classified using the International Classification of Diseases (ICD) versions eight (1969−1986), nine (1987−1996), or ten (1997−present). A parent was considered to have an MD if at least one of the following diagnoses had been assigned: substance use disorders, disorders with psychotic features, mood disorders, anxiety disorders, eating disorders, nonorganic sleep disorders, personality disorders, mental retardation, developmental disorders, and conduct disorders (specific ICD codes are presented in Supplement 1, available online). In addition, the diagnosis had to be assigned before the child's 10th birthday. Prevalence of mothers and fathers within various disorder categories are presented in Tables S2 and S3, respectively, available online.

At both follow-ups, disruptive behavior subsamples had significantly higher prevalences of maternal mental disorder (follow-up 15 years: 6.4% versus 5.0%; χ^2 [1, n = 6,319] = 5.43, $p < .05$; 18 years: 5.4% versus 4.5%; χ^2 [1, n = 3,068] = 4.25, $p < .05$) and paternal mental disorder (follow-up 15 years: 5.7% versus 4.0%; χ^2 [1, n = 6,319] = 10.20, $p < .01$; 18 years: 6.2% versus 3.7%; χ^2 [1, n = 3,068] = 4.75, $p < .05$) compared to nondisruptive behavior samples (follow-up 15 years: n = 4,104; 18 years: n = 1,878).

Parental Education. The educational level of each parent was obtained during the telephone interview at baseline. First, education level was coded into three different categories: 1 (completed primary school or less [≤9 years of formal education]); 2 (completed a high school education

TABLE 1 Descriptive Statistics for Children With Complete Data at Baseline and Age 15 Years

Variable Function and Child Age at Assessment	Variable	Total Sample (N = 6,319) Mean (SD)[a]	Total Sample (N = 6,319) Range	Disruptive Behavior Subsample (n = 2,215) Mean (SD)[a]	Disruptive Behavior Subsample (n = 2,215) Range
Predictors at 9 y	Disruptive behavior (PR)	0.62 (1.34)	0–21	1.78 (1.75)	0.5–21
	Maternal mental disorder (Reg.) [n (%)]	345 (5.5%)	0–1	141 (6.4%)	0–1
	Paternal mental disorder (Reg.) [n (%)]	290 (4.6%)	0–1	127 (5.7%)	0–1
Covariates at 9 y	Parental education level (PR)	4.80 (1.00)	2–6	4.13 (1.00)	2–6
	Maternal age at childbirth (PR)	31.02 (4.52)	16–50	30.82 (4.47)	16–46
	Paternal age at childbirth (PR)	33.45 (5.74)	17–65	33.33 (5.75)	18–65
	Child's sex, male (PR) [n (%)]	2885 (45.7%)	0–1	1120 (50.6%)	0–1
Outcomes at 15 y	Nonviolent crime (SR)	0.57 (1.86)	0–52	0.70 (1.94)	0–37
	Violent crime (SR)	0.45 (1.23)	0–36	0.48 (1.29)	0–20
	Proactive aggression (SR)	0.70 (1.53)	0–24	0.87 (1.74)	0–16
	Reactive aggression (SR)	4.55 (3.50)	0–22	5.21 (3.80)	0–22
	Truancy (SR)	0.52 (0.98)	0–4	0.65 (1.12)	0–4
	Frequent alcohol consumption (SR) [n (%)]	1046 (16.6%)	0–1	387 (17.5%)	0–1
	Frequent alcohol intoxication (SR)	1.09 (1.53)	0–5	1.16 (1.58)	0–5
	Conduct problems (PR)	1.73 (1.43)	0–10	2.02 (1.56)	0–10
	Emotional problems (PR)	2.85 (2.23)	0–10	2.96 (2.33)	0–10
	Peer problems (PR)	1.76 (1.57)	0–9	1.96 (1.65)	0–9
	Prosocial behavior (PR)	7.06 (1.69)	0–10	6.89 (1.79)	0–10
	Cumulative risk index [n (%)]		0–10		0–10
	0 Poor outcomes	1391 (22.0)		380 (17.2)	
	1 Poor outcome	1488 (23.5)		452 (20.4)	
	2 Poor outcomes	1142 (18.1)		386 (17.4)	
	3 Poor outcomes	796 (12.6)		304 (13.7)	
	≥4 Poor outcomes	1502 (23.8)		693 (31.3)	

Note: *PR = parent-reported; Reg. = registry; SR = self-reported.*
[a]*Data are Mean (SD) unless otherwise noted.*

[10–12 years]); and 3 (university studies or equivalent [≥13 years]). Next, educational level of both parents were summed, resulting in a score ranging from 2 to 6. If information about the education of one parent was missing, the educational level of the other parent with available data was imputed.

Outcome Measures at Age 15 Years

Information was collected on various outcomes at age 15 years, relying on self- and parent-reports. Reactive (or impulsive) and proactive (or planned) aggression was assessed through a youth self-report questionnaire.[16] Criminality was assessed with a self-report tool that assessed the frequency of violent and nonviolent criminal acts.[17] Conduct problems of the child were assessed using the Conduct Problems subscale of the Strengths and Difficulties Questionnaire (SDQ) parent version.[18] Alcohol misuse was measured through self-report,[19] and refers to frequent alcohol consumption and/or frequent alcohol intoxication. Emotional problems, peer problems, and low prosocial behavior were measured by means of the corresponding scales of the SDQ parent version. In line with prior work,[20] self-reported truancy of the child was assessed using one item ("Did you ever skip school?"). Details of these measures (including example items) are provided in Supplement 2, available online.

Outcome Measures at Age 18 Years

Information was collected on various outcomes at age 18 years, thereby, relying on self- and parent-reports, and a registry. Aggression was assessed using self-report[21] and parent-report questionnaires.[22] Criminality was assessed with the same self-report tool that was used at age 15 to assess the frequency of violent and nonviolent criminal acts.

TABLE 2 Descriptive Statistics for Children With Complete Data at Baseline and Age 18 Years

Variable Function and Child Age at Assessment	Variable	Total Sample (N = 3,068)		Disruptive Behavior Sample (n = 1,190)	
		Mean (SD)[a]	Range	Mean (SD)[a]	Range
Predictors at 9 y					
	Disruptive behavior (PR)	0.72 (1.43)	0–13	1.85 (1.79)	0.5–13
	Maternal mental disorder (Reg.) [n (%)]	159 (5.2%)	0–1	64 (5.4%)	0–1
	Paternal mental disorder (Reg.) [n (%)]	134 (4.4%)	0–1	74 (6.2%)	0–1
Covariates at 9 y					
	Parental education level (PR)	4.80 (1.01)	2–6	4.78 (1.01)	2–6
	Maternal age at childbirth (PR)	30.68 (4.59)	19–56	30.46 (4.67)	16–46
	Paternal age at childbirth (PR)	33.05 (5.62)	16–46	32.77 (5.59)	19–56
	Child's gender male (PR) [n (%)]	1,331 (43.4%)	0–1	584 (49.1%)	0–1
Outcomes at 18 y					
	Nonviolent crime (SR)	1.57 (3.18)	0–37	2.02 (3.76)	0–37
	Violent crime (SR)	0.95 (1.97)	0–20	1.27 (2.32)	0–20
	Aggression (SR)	6.62 (5.07)	0–25	7.67 (5.39)	0–25
	Consequences of antisocial behavior (SR)	0.51 (1.32)	0–14	0.71 (1.64)	0–14
	Truancy (SR)	1.45 (1.48)	0–4	1.68 (1.53)	0–4
	Alcohol misuse (SR)	4.74 (4.16)	0–34	5.12 (4.51)	0–34
	Rule-breaking behavior (PR)	13.68 (1.73)	6–34	14.02 (2.19)	6–34
	Aggression (PR)	18.60 (3.01)	8–40	19.40 (3.51)	8–34
	Emotional problems (PR)	16.01 (3.21)	7–39	16.57 (3.65)	7–39
	School performance (Reg.)	236.11 (55.32)	0–320	228.15 (56.91)	0–320
	Cumulative risk index [n (%)]		0–10		0–10
	0 Poor outcomes	423 (13.8)		105 (8.8)	
	1 Poor outcome	622 (20.3)		166 (13.9)	
	2 Poor outcomes	562 (18.3)		207 (17.4)	
	3 Poor outcomes	438 (14.3)		185 (15.5)	
	≥4 Poor outcomes	1,023 (33.3)		527 (44.3)	

Note: PR = parent-reported; Reg. = registry; SR = self-reported.
[a]*Data are Mean (SD) unless otherwise noted.*

Rule-breaking behavior was assessed by the parent-reported Adult Behavior Checklist (ABCL).[22] Consequences of antisocial behavior was assessed through a self-report questionnaire that taps social consequences (eg, reprimands) caused by involvement in antisocial behavior.[21] The self-report Alcohol Use Disorders Identification Test (AUDIT)[23] was used to assess alcohol consumption, drinking behavior (dependence), and alcohol-related problems. Gender-specific AUDIT cut-offs were used to define Alcohol Misuse. Emotional problems were assessed by the parent-reported Anxious/Depressed subscale of the aforementioned ABCL. Truancy was assessed as described earlier (Outcome Measures at Age 15). Registered school performance of the child was assessed using the sum of the final grades of 16 subjects (eg, mathematics, English) in primary school. The grades were obtained through the National School Registry. Details of these measures are given in Supplement 2, available online.

Cumulative Poor Functioning at Ages 15 and 18 Years

For each follow-up assessment, a cumulative risk index was computed by summing the times that a child was above the cut-off used to define poor outcomes (see Statistical Analyses). The score for this index ranged from "0" (indicating that the child did not experience any of the poor outcomes measured at follow-up) to "10" (indicating that the child experienced all 10 poor outcomes measured at follow-up) (for prevalences, see Tables 1 and 2). At both follow-ups, disruptive behavior subsamples had significantly higher prevalences of maternal mental disorder (follow-up 15 years: 6.4% versus 5.0%; χ^2 [1, n = 6,319] = 5.43, $p < .05$; 18 years: 5.4% versus 4.5%; χ^2 [1, n = 3,068] = 4.25,

$p < .05$) and paternal mental disorder (follow-up 15 years: 5.7% versus 4.0%; χ^2 [1, n = 6,319] = 10.20, $p < .01$; 18 years: 6.2% versus 3.7%; χ^2 [1, n = 3,068] = 4.75, $p < .05$) compared to nondisruptive behavior samples (follow-up 15 years: n = 4,104; 18 years: n = 1,878).

Statistical Analyses

The continuous outcome variables were substantially skewed, even after data normalization transformations. Therefore, consistent with a large body of research,[24,25] dichotomized outcome variables were used. Specifically, echoing prior work,[24,26] all outcome measures, except self-reported crime, were dichotomized into high (ie, the 30% highest scores, 1, which is indicative of low functioning) versus low (ie, 70% lowest scores, 0). These cutoffs were also used because Swedish norms were unavailable for the majority of the outcome measures. Because high scores on prosocial behavior and grades indicate a high level of functioning, these were dichotomized differently, with a low level of functioning corresponding with the 30% lowest scores (indicated by a score of 1), and high functioning corresponding with the 70% highest scores (score of 0). Table S4, available online, describes with which raw scores the dichotomization cutpoints correspond. In line with prior research on the prediction of criminal outcomes,[27,28] we used dichotomized variables (no offenses versus one or more offenses) to define future violent and nonviolent criminality.

Generalized linear mixed models (GLMMs) for logistic regression, unless otherwise specified, were conducted, using a binomial distribution with a logit link. GLMMs combine both linear mixed models and generalized linear models, and enable the introduction of random effects. The introduction of a random effect (ie, twins nested within families) is needed in this study to correct for dependency of observations (ie, one parent reporting on the behavior of two twins). In this study, a robust estimator (Huber/White/sandwich estimation) was used to estimate the covariance. This estimator corrects for the dependence of observations and other departures from normality, such as under- and overdispersion. Wald χ^2 tests were used to test the fixed effects. For the fixed effects corresponding odds ratios (ORs) and 95% confidence intervals (CIs) were computed and reported.

Poisson or negative binomial models can accommodate nonnormality without having to resort to dichotomizing outcomes. However, the appropriateness of these models varied across outcome measures. Therefore, logistic regression models also helped to test all outcome measures uniformly. Yet, when appropriate, we ran negative binomial and/or Poisson regression analyses and found that the pattern of the findings was substantially similar to the results of the logistic regression analyses.

Specifically, using GLMM for logistic regression, four models were tested. The first model was a crude effects model consisting of child DB (continuous), paternal MD (dichotomous), or maternal MD (dichotomous), together with four theoretically relevant control variables: parental education level,[29] maternal age at childbirth,[30] paternal age at childbirth,[31] and sex of the child.[32] In the second model, child DB, paternal MD, and maternal MD were included simultaneously in an adjusted model, together with the aforementioned control variables. These latter two models were run in the total sample to assess the influence of parental MD and child DB in middle and late adolescence. However, to test whether parental MD is a risk factor for future maladjustment among children who already display DB, both models (refered to as model 3 and 4, respectively) were repeated in a subsample of children who displayed at least some DB (ie, a raw DB score of 0.5 or higher). Of note, GLMMs were conducted separately for individuals with outcome data at age 15 years (n = 6,319; DB sample n = 2215, 35.1% of total sample at age 15) and for individuals with outcome data at age 18 years (n = 3,068; elevated DB sample n = 1,190, 38.8% of total sample at age 18) for two reasons. First, there was a relatively low number of children for whom data were available for both follow-up assessments (n = 1,696), and only 126 of these children had a parent with an MD. Therefore, it was not tenable to run the GLMMs. Second, different outcome measures were used across the follow-up assessments, limiting the possibility of testing stability and change from age 15 to age 18 without introducing measurement bias. When using CD and ODD symptoms as separate predictors instead of combining CD and ODD in an omnibus variable (ie, DB), results remained substantially similar. Details are available upon request from the first author. The analyses were performed in SPSS version 23, using the IBM SPSS MIXED function. We used $p < .05$ as an indicator of statistical significance. Sequential Bonferroni was used to adjust for multiple comparisons. Two-tailed tests were used in all analyses.

Attrition

At age 15 years, there were 1,680 of 7,999 children who were not included in the analyses because of some degree of missing data. These children did not differ from children without missing data at age 15 years in terms of maternal and paternal age at childbirth. However, children with (versus without) missing data were more often boys (45.3% versus 60.2%, $p < .001$) and had parents with lower educational levels ($p < .001$). At age 18 years, 3,305 of 6,373 children were excluded because of missing data. Significant differences emerged between children with and without missing data in terms of age of the mother at birth (mean = 30.4, SD = 4.72 versus 30.7 years,

SD = 4.56, $p = .023$, $d = 0.07$), percentage of boys (48.3% versus 60.2%, $p < .001$), and parental education level ($p < .001$), but not in terms of paternal age at childbirth.

RESULTS

Outcomes at Age 15 Years

Total Sample. First, crude models were run for the predictors, child DB, paternal MD, and maternal MD separately (Table 3, model 1). Child DB was significantly positively associated with all outcome measures at age 15 years ($p < .01$). Paternal MD was significantly positively associated with self-reported violent crime, nonviolent crime, reactive aggression, truancy, and the cumulative risk index. Maternal MD was significantly positively related to parent-reported peer problems and self-reported truancy.

Next, adjusted models in which all three predictors were included simultaneously (Table 3, model 2) showed that child DB remained significantly associated with all outcomes. Paternal MD remained significantly positively related to violent crime, nonviolent crime, truancy, and the cumulative risk index, although the prospective association with reactive aggression was no longer statistically significant. Maternal MD remained positively associated with peer problems but not to truancy at age 15.

Subsample of Children With Disruptive Behavior. Paternal MD was not predictive of any of the outcomes, whereas maternal MD was positively associated with peer problem in both the crude model (OR = 1.64; 95% CI = 1.13; 2.38) and the adjusted model (OR = 1.62; 95% CI = 1.12; 2.34). For details, see Table S5, available online.

Outcomes at Age 18 Years

Total Sample. Child DB was associated with all outcomes at age 18 years ($p < .01$) in the crude model (Table 4). Paternal MD was positively associated with self-reported nonviolent crime, aggression, consequences of antisocial behavior, truancy, registered school performance, and the cumulative risk index of poor functioning. Maternal MD was significantly positively related to only one outcome, namely, parent-reported rule-breaking behavior. These prospective relations between paternal MD and future outcomes remained significant in the adjusted models (model 2), although maternal MD was no longer related to rule-breaking behavior in model 2.

Subsample of Children With Disruptive Behavior. Paternal MD was prospectively related to self-reported nonviolent crime, consequences of antisocial behavior, truancy, registered poor school performance, and the cumulative risk index, both in the crude (model 3) and adjusted models (model 4) (Table 5). Maternal MD was not predictive of any of the outcomes in models 3 and 4. It could also be the case that the "what" question (ie, Is there an internalizing or externalizing MD present in the parents?) might be more important than the "who" question (ie, Does the mother or the father have an MD?), especially because a higher prevalence of externalizing disorders in fathers than in mothers might explain why paternal MD was most often related to the reported antisocial outcomes in the subsample of children with DB at age 18. We addressed this issue in Supplement 3, and in Tables S6 and S7, available online. In short, the outcomes of these analyses suggest that the "what" and "who" questions are equally important.

DISCUSSION

The main aim of this study was to test whether 9-year-old children with disruptive behavior (DB) who have a parent with a mental disorder (MD) display increased maladjustment in adolescence compared to children with DB whose parents were without an MD. Overall, the current findings partially support this hypothesis. Specifically, maternal MD was a risk factor for peer problems at age 15, a finding that adds to prior work showing that maternal MD, but not paternal MD, was predictive of reduced social skills in preschoolers with DB.[13] This difference could be explained by gender-specific parenting behavior, with fathers tending to focus on promoting their child's exploratory behavior and rough-and-tumble play, and mothers more focused on social–affective behavior.[33] Furthermore, paternal MD was a risk factor for children with DB to show higher levels of nonviolent crime and truancy, to experience more negative consequences of antisocial behavior (eg, school suspensions), to perform worse at school at age 18, and to experience multiple poor outcomes (cumulative risk index). Importantly, nonviolent crime in adolescence has been demonstrated to be a risk factor for reoffending,[34] whereas truancy in adolescence is also a risk factor for later crime,[35] mental health problems,[36] and academic underachievement.[37] In addition, poor school performance in adolescence increases the risk of later health problems,[38] reliance on government assistance, illicit substance use, arrest, and being fired.[39] Therefore, paternal MD may not only jeopardize the transition from childhood to adolescence (this study), but also a succesfull transition from adolescence to adulthood, a possibility that is in need of empirical evaluation.

Despite the aforementioned findings in partial support of our hypothesis, it cannot be disregarded that in children with DB, parental MD was more often unrelated to the majority of outcomes at ages 15 and 18, including the ones

TABLE 3 Odds Ratios and 95% CIs of the Fixed Part of the Crude and Adjusted Main Effects Models at Follow-up at 15 Years

		Criminality		Aggression		Problems						
		Violent (SR)	Nonviolent (SR)	Proactive (SR)	Reactive (SR)	Conduct (PR)	Emotional (PR)	Peer (PR)	Alcohol (SR)	Truancy (SR)	Low Prosocial (PR)	Cumulative Risk[a]
Predictor	Model	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Child DB	1	**1.19**** (1.13–1.25)	**1.18**** (1.07–1.17)	**1.15**** (1.10–1.21)	**1.24**** (1.18–1.30)	**1.23**** (1.17–1.29)	**1.11**** (1.06–1.17)	**1.17**** (1.12–1.22)	**1.07**** (1.03–1.12)	**1.14**** (1.08–1.19)	**1.08**** (1.03–1.13)	**1.10**** (1.08–1.11)
	2	**1.19**** (1.13–1.25)	**1.12**** (1.06–1.17)	**1.15**** (1.10–1.20)	**1.24**** (1.18–1.30)	**1.23**** (1.17–1.29)	**1.11**** (1.06–1.16)	**1.17**** (1.12–1.22)	**1.07**** (1.03–1.12)	**1.13**** (1.08–1.19)	**1.08**** (1.03–1.13)	**1.09**** (1.08–1.11)
Paternal MD	1	**1.65**** (1.22–2.23)	**1.40*** (1.04–1.89)	1.18 (0.83–1.67)	**1.38*** (1.02–1.87)	1.28 (0.96–1.70)	1.16 (0.85–1.57)	1.10 (0.83–1.45)	1.31 (0.96–1.79)	**1.67**** (1.26–2.21)	0.96 (0.70–1.32)	**1.19**** (1.06–1.34)
	2	**1.59**** (1.16–2.17)	**1.36*** (1.00–1.84)	1.12 (0.78–1.60)	1.29 (0.94–1.78)	1.19 (0.89–1.61)	1.11 (0.82–1.50)	1.02 (0.77–1.34)	1.29 (0.94–1.77)	**1.58**** (1.19–2.11)	0.93 (0.67–1.29)	**1.17*** (1.03–1.30)
Maternal MD	1	0.84 (0.60–1.19)	0.98 (0.75–1.29)	1.09 (0.78–1.53)	1.10 (0.84–1.44)	1.09 (0.84–1.42)	1.16 (0.87–1.56)	**1.41**** (1.10–1.80)	0.97 (0.72–1.31)	**1.33*** (1.01–1.74)	1.05 (0.78–1.41)	1.06 (0.95–1.19)
	2	0.77 (0.54–1.09)	0.93 (0.71–1.22)	1.04 (0.74–1.46)	1.02 (0.77–1.35)	1.02 (0.78–1.33)	1.12 (0.83–1.51)	**1.36*** (1.06–1.73)	0.94 (0.70–1.26)	1.25 (0.95–1.64)	1.03 (0.78–1.38)	1.02 (0.92–1.14)

Note: *N = 6,319. Model 1 = crude model including the control variables: sex of child, parental education level, maternal age at birth, and paternal age at birth; model 2 = adjusted model including child DB, paternal MD, maternal MD, and the same control variables as in model 1. Boldface type indicates statistical significance. DB = disruptive behavior; MD = mental disorder; OR = odds ratio; PR = parent-reported; SR = self-reported.*

[a]*Cumulative risk was predicted through negative binomial regressions.*

$^{*}p < .05$; $^{**}p < .01$.

TABLE 4 Odds Ratios and 95% CIs of the Fixed Part of the Crude and Adjusted Main Effects Models at Follow-up at 18 Years

		Criminality		Aggression		Problems						
		Violent (SR)	Nonviolent (SR)	Aggression (SR)	Aggression (PR)	Conseq. of Antisocial Behavior (SR)	Rule Breaking (PR)	Emotional (PR)	Alcohol (SR)	Truancy (SR)	Poor School Performance (Reg.)	Cumulative Risk[a]
Predictor	Model	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Child DB	1	1.22**	1.13**	1.22**	1.51**	1.14**	1.33**	1.30**	1.08*	1.19**	1.16**	1.12**
		(1.15, 1.30)	(1.06, 1.21)	(1.15, 1.29)	(1.41, 1.62)	(1.08, 1.21)	(1.25, 1.42)	(1.23, 1.39)	(1.01, 1.14)	(1.12, 1.27)	(1.09, 1.24)	(1.11, 1.14)
	2	1.22**	1.13**	1.22**	1.51**	1.14**	1.33**	1.30**	1.07**	1.18**	1.16**	1.12**
		(1.15, 1.30)	(1.07, 1.21)	(1.14, 1.29)	(1.40, 1.62)	(1.07, 1.21)	(1.24, 1.42)	(1.22, 1.39)	(1.01, 1.14)	(1.11, 1.26)	(1.08, 1.24)	(1.10, 1.14)
Paternal MD	1	1.44	1.87**	1.72**	1.45	1.85**	1.17	1.01	1.38	2.71**	1.89**	1.34**
		(0.96, 2.15)	(1.23, 2.83)	(1.14, 2.59)	(0.94, 2.24)	(1.24, 2.77)	(0.68, 2.03)	(0.70, 1.73)	(0.88, 2.16)	(1.72, 4.26)	(1.20, 2.99)	(1.16, 1.55)
	2	1.38	1.90**	1.68*	1.33	1.77**	1.01	0.99	1.34	2.60**	1.76*	1.29**
		(0.92, 2.07)	(1.25, 2.89)	(1.10, 2.58)	(0.82, 2.16)	(1.18, 2.66)	(0.57, 1.79)	(0.61, 1.59)	(0.86, 2.11)	(1.66, 4.07)	(1.11, 2.79)	(1.12, 1.49)
Maternal MD	1	1.12	0.83	0.99	1.20	1.25	1.66*	1.44	1.14	1.32	1.44	1.13
		(0.75, 1.68)	(0.54, 1.29)	(0.63, 1.55)	(0.83, 1.73)	(0.80, 1.95)	(1.04, 2.64)	(0.97, 2.13)	(0.71, 1.84)	(0.80, 2.19)	(0.86, 2.41)	(0.97, 1.31)
	2	0.98	0.72	0.84	0.96	1.09	1.45	1.28	1.07	1.09	1.26	1.03
		(0.65, 1.48)	(0.46, 1.13)	(0.52, 1.34)	(0.64, 1.44)	(0.70, 1.71)	(0.89, 2.37)	(0.85, 1.93)	(0.66, 1.73)	(0.66, 1.80)	(0.75, 2.12)	(0.89, 1.20)

Note: *N = 3,068. Model 1 = crude model including the control variables: sex of child, parental education level, maternal age at birth, and paternal age at birth; model 2 = adjusted model including child DB, paternal MD, maternal MD, and the same control variables as in model 1. Boldface type indicates statistical significance. Conseq. = consequences; DB = disruptive behavior; MD = mental disorder; OR = odds ratio; PR = parent-reported; Reg. = registry; SR = self-reported.*

[a]*Cumulative risk was predicted through negative binomial regressions.*

$^{*}p < .05$; $^{**}p < .01$.

TABLE 5 Odds Ratios and 95% CIs of the Fixed Part of the Crude and Adjusted Main Effects Models at Follow-up at 18 Years in a Subsample of Children With Disruptive Behavior

		Criminality		Aggression		Problems						
		Violent (SR)	Nonviolent (SR)	Aggression (SR)	Aggression (PR)	Conseq. of Antisocial Behavior (SR)	Rule Breaking (PR)	Emotional (PR)	Alcohol (SR)	Truancy (SR)	Poor School Performance (Reg.)	Cumulative Risk[a]
Predictor	Model	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Paternal MD	3	1.59 (0.93–2.71)	2.03* (1.07–3.84)	1.51 (0.92–2.47)	1.13 (0.67–1.90)	2.24** (1.30–3.87)	1.15 (0.59–2.25)	0.90 (0.49–1.64)	1.75 (0.96–3.21)	2.40** (1.33–4.33)	2.02* (1.11–3.67)	1.30** (1.12–1.51)
	4	1.50 (0.87–2.58)	2.05* (1.08–3.90)	1.53 (0.91–2.56)	1.11 (0.65–1.89)	2.13** (1.22–3.72)	1.12 (0.58–2.17)	0.82 (0.45–1.51)	1.70 (0.93–3.11)	2.22* (1.21–4.08)	2.01* (1.09–3.72)	1.28** (1.09–1.49)
Maternal MD	3	1.55 (0.91–2.65)	1.04 (0.55–1.98)	0.98 (0.54–1.78)	1.11 (0.71–1.75)	1.57 (0.85–2.90)	1.24 (0.69–2.24)	1.63 (0.96–2.76)	1.33 (0.70–2.53)	1.87 (0.96–3.64)	1.13 (0.56–2.31)	1.18 (1.00–1.39)
	4	1.46 (0.85–2.53)	0.94 (0.49–1.80)	0.92 (0.49–1.71)	1.10 (0.69–1.74)	1.40 (0.75–2.62)	1.22 (0.68–2.19)	1.68 (0.97–2.89)	1.23 (0.65–2.33)	1.67 (0.83–3.33)	1.03 (0.49–2.16)	1.14 (0.96–1.36)

Note: *n = 1,190. Model 1 = crude model including the control variables: sex of child, parental education level, maternal age at birth, and paternal age at birth; model 2 = adjusted model including paternal MD, maternal MD, and the same control variables as in model 1. Conseq. = consequences; MD = mental disorder; OR = odds ratio; PR = parent-reported; Reg. = registry; SR = self-reported.*

[a]*Cumulative risk was predicted through negative binomial regressions.*

*$p < .05$; **$p < .01$.

that most clearly affect society as a whole, namely, aggression and violent criminality. Intriguingly, in the total sample, main effects were revealed for paternal MD as predictor of these latter two indices of severe antisocial behavior, suggesting that parental MD has more prognostic value if one does not specifically focus on 9-year-old children who already display DB. Although replication is warranted, we should note that these findings dovetail with prior work in criminology showing that well-established risk factors for first-time offending are less useful to predict reoffending.[40] Also, it should be noted that most prospective associations between parental MD and poor outcomes, including the cumulative risk index, in children with DB were not significant at age 15 but were significant at age 18. It may be that children at age 18 become increasingly independent of their parents in various areas of life. This developmental transition increases differences between individuals (which may be reflected in the larger standard deviation in outcome measures at age 18 than at age 15; Tables 1 and 2), and therefore also increases the likelihood of finding significant associations in late as compared to middle adolescence.

Notwithstanding that this study's main focus was on the effects of parental MD among children with DB, our findings also contribute substantially to the literature on outcomes of children of parents with an MD. Crucially, fathers have been understudied compared to mothers in studies linking parental MD and child maladjustment.[13] The current investigation provides evidence that paternal MD is predictive of various outcomes at age 15 and 18, suggesting that mothers are not the only parent of interest when examining the prognostic value of parental MD. In fact, maternal MD merely predicted increased peer problems at age 15 years, a finding that is surprising in light of evidence that maternal MD is a risk factor for a variety of poor psychosocial outcomes in offspring.[41] Nevertheless, there is some prior evidence to suggest that paternal MD is a stronger risk factor for emotional and behavioral problems in older children and adolescents compared to younger children, whereas maternal MD has a larger impact on younger children.[4] Also, prior work suggests that paternal MDs are more strongly associated with child behavioral problems than child internalizing problems.[42,43] This is consistent with our results showing that paternal MD in the total sample was associated with increased rates of antisocial behavior (eg, aggression, crime, and truancy) but not once to emotional problems. Furthermore, our findings are also consistent with evidence that fathers have a larger effect than mothers on the development of delinquency in their offspring,[44] and suggest that the impact of paternal MD extends well into middle and late adolescence.

This study also contributes to the broader literature on early-onset DB, generally showing that conduct problems in children younger than 10 years[10,45] are significant predictors of antisocial behavior, psychopathology, and school drop-out in adolescence and adulthood. The current study confirmed these findings, with early-onset DB predicting worse outcomes on all measures at age 15 and 18 years, even after controlling for paternal and maternal MD. Importantly, parent-reported DB was not only predictive of parent-reported outcomes (eg, conduct problems, emotional problems, and low prosocial behavior) at both follow-up intervals but also of negative outcomes that were based on youth self-report (eg, proactive aggression, violent criminality, and alcohol use) and information from registries (ie, poor school performance), showing that the prognostic usefulness of child DB was not solely caused by shared-method variance. To bolster what is known about parental MD as a predictor of poor outcomes in children with DB, we focused on the presence of DB. Consequently, the vast majority of children with DB in our subsample likely do not display severe DB and do not meet criteria to warrant a formal ODD or CD diagnosis. Future research is warranted to see whether our findings can be replicated in children with severe DB or with formal disruptive behavior disorder diagnoses.

This study has several strengths, including its longitudinal design and the use of a well-described sample, the availability of multiple information sources, and the variety of outcomes across various domains that were assessed at two different follow-ups.[14] However, the findings should be interpreted in the context of various limitations. First, the present study used lifetime prevalence of parental MD and child DB, which implies that it is uncertain whether parental MD occurred before, at the same time, or after the onset of child DB. This might have hampered the likelihood of finding significant main effects of parental MDs in children with DB, especially as there is some evidence that the timing of exposure of parental MD matters when studying outcomes in children.[46] Second, specific mental disorders in parents have been associated with different child outcomes,[4,47] and there is some evidence to suggest that relations between specific parental mental disorders with future child outcomes are influenced by the sex of the parent.[13] Notwithstanding that our exploratory post hoc analyses (with the broad disorder categories externalizing and internalizing disorder as predictors) support these findings to some extent, prevalence issues hampered us from testing the effect of specific MDs (eg, substance use disorder, major depressive disorder) in parents in general, or in mothers and fathers seperately. Also, regardless of the large number of children and parents included in this study, the

number of children with DB who had parents with an MD was rather low. Thus, prevalence issues may also have hampered the likelihood of finding significant effects of parental MDs in children with DB, whereas interactions between maternal and paternal MD were not possible to study because the number of dyads in which both parents were diagnosed with an MD ranged from 14 (subsample with DB at age 18) to 39 (total sample at age 15). Third, even though statistical testing for continuous outcomes was not an option (see Method section), it can be argued that dichotomizing our outcome variables may have decreased the power to reveal significant prospective relations. However, the use of logistic regression and dichotomization of outcome variables has benefits as well,[48] and enhances comparison with prior work that used distribution-based cut-offs.[24-26] Fourth, officially recorded parental mental disorders are most likely an underestimation of the true extent of parental mental disorders, suggesting that future research also needs to rely on diagnostic interviews with parents.

This study supports the importance of treating parental MD, in both children with and without early-onset DB. Although screening for parental MD in the general adult population would be time consuming and costly, relatively easy gains could be made in children who are already in treatment for DB. Treatment-as-usual of the child could be augumented with additional screening and, if necessary, treatment of mental health problems in the child's parents, as improvement of parental mental health has been associated with better child outcomes.[49] Furthermore, the current study clearly underscores the urgency of screening for early-onset child DB in the community, as an accurate identification of these children may eventually increase the likelihood that intervention programs might mitigate or even prevent a developmental pathway toward disruptive behavior disorders and maladjustment in adolescence and adulthood.[50]

In conclusion, the results strongly suggest that fathers must be considered when studying prospective associations between parental MD and offspring psychosocial functioning. Crucially, we provided novel evidence that children with early-onset DB who had a parent with (versus without) a registered MD were at an increased risk for poor psychosocial functioning in middle and late adolescence.

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