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Facts of aggression

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CHAPTER 2

CHILDREN WITH EARLY-ONSET DISRUPTIVE BEHAVIOR: PARENTAL MENTAL DISORDERS PREDICT POOR PSYCHOSOCIAL FUNCTIONING IN ADOLESCENCE

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ABSTRACT

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*.

Methods: Parents of 9-year-old children reported on child DB, while a patient registry was used to determine parental MD. At follow-ups at 15 (N = 6319) and 18 years (N = 3068) 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; $ps < .01$), 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; $ps < .05$). In the subsample of children with DB, paternal MD predicted criminality, consequences of antisocial behavior, truancy, poor school performance, and cumulative risk, while maternal MD predicted peer problems (mean OR = 1.94; range = 1.30-2.40; $ps < .05$).

Conclusion: This study provides novel evidence that parental MD puts 9-year-olds with DB at risk for negative outcomes in adolescence. Additionally, 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.

Lay summary: Children with behavioral problems experience on average more negative long-term outcomes than typically developing children, and more often have parents with psychiatric disorders. In this study we showed that Swedish twins who had behavioral problems and parents with a psychiatric disorder were more likely to experience bad outcomes in adolescence, such as criminal behavior and poor academic performance, than twins with behavioral problems only. Interestingly, paternal psychiatric disorders seemed far more predictive of negative outcomes than maternal psychiatric disorders. These findings suggest

that taking parents into account when treating children with behavioral problems would be promising for clinical practice and treatment effectiveness.

INTRODUCTION

It is well documented that children with early-onset disruptive behavior (DB), including oppositional defiant and conduct disorder symptoms, have a high risk for adverse psychosocial outcomes in adolescence and adulthood, such as school dropout, criminality, substance abuse, reduced social skills, and mental health problems (Baker, 2016; Bevilacqua, Hale, Barker, & Viner, 2017; Cleary & Nixon, 2012). There are also clear indications that parental mental disorders (MDs) are involved in the onset (Connell & Goodman, 2002) and maintenance (DeKlyen, Bierbaum, Speltz, & Greenberg, 1998; Odgers et al., 2007) of childhood DB and other negative psychosocial outcomes (Flouri & Ioakeimidi, 2017). Children with DB often cause emotional distress and discord in the family (George, Herman, & Ostrander, 2006), suggesting that childhood DB increase the risk for mental disorder in parents (Panico, Becares, & Webb, 2014). However, it is not well researched if parental MD increases the risk for 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 (Wertz et al., 2018) or did not test the prognostic usefulness of parental MD in a subsample of youth with DB (Network & Arsenio, 2004). This lack of research is surprising since parental MDs have been considered to constitute a major risk factor for treatment failure of childhood DB (Shelleby & Kolko, 2015).

We are aware of only one study that has addressed this topic. In a sample of 132 3-year-old preschoolers with DB, Breaux and colleagues (2014) showed that indices of maternal and paternal psychopathology were predictive of parent-ratings of child externalizing and internalizing problems and social skill deficits three years later (Breaux, Harvey, & Lugo-Candelas, 2014). These findings suggest that parental MD in children with DB is a risk factor of poor prognoses. Yet, the Breaux study (Breaux et al., 2014) 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 to reveal 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 three-year follow-up interval to study outcomes of preschoolers with a MD. Therefore, it is uncertain if 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 if 9-year-old children with DB are at a greater risk for maladjustment in middle (age 15) and late adolescence (age 18) 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 (Wertz et al., 2018), we examined if child DB and parental MD predicted each outcome separately as well as a cumulative index of poor functioning.

METHODS

Participants

The Child and Adolescent Twin Study in Sweden (CATSS) is a nation-wide longitudinal study that targets all twins born in Sweden since July 1992 (Anckarsäter et al., 2011). Parents of twins were administered the Autism-Tics, AD/HD and other Comorbidities inventory (A-TAC) by telephone in connection with the twin's ninth birthday (twins born from July 1, 1992 to June 30, 1995 were included at age 12). 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 onwards, while the follow-up at 18 years includes twins born in 1992 and onwards. 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 8906 twins (born 1992-1999), of which 7105 participated at the first follow-up, and 4492 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 6319 children, and for whom outcome measures of interest were available at age 18 (second follow-up), resulting in a sample of 3068 children. A subsequent selection of children with DB resulted in a subsample of 2215 children at the first follow-up, and a subsample of 1190 children at the second follow-up. Descriptive information of all samples can be retrieved from Tables 1 and 2.

Baseline measures at Age 9

Parent-reported disruptive behavior (DB)

DB of the child was assessed using A-TAC, which consists of 96 questions covering common child and adolescent psychiatric disorders, including oppositional defiant disorder (ODD) and conduct disorder (CD; Hansson et al., 2005). 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 can be retrieved from 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. MDs 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 a 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 tenth birthday. Prevalence of mothers and fathers within various disorder categories are presented in Tables S2 and S3, respectively, and are available online.

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 (10-12 years)) and 3 (university studies or equivalent (≥ 13 years)). Next, education 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 education level of the other parent with available data was imputed.

Outcome measures at age 15

Information was collected on various outcomes at age 15, relying on self- and parent-reports. *Reactive* (or impulsive) and *proactive* (or planned) *aggression* was assessed through a youth self-report questionnaire (Raine et al., 2006). *Criminality* was assessed with a self-report tool that assessed the frequency of violent and non-violent criminal acts (Ring, 1999). *Conduct problems* of the child were assessed using the Conduct Problems subscale of the Strengths and Difficulties Questionnaire's (SDQ) parent version (Goodman, 1997). *Alcohol misuse* was measured through self-report (Englund, 2016), 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 (Norén Selinus et al., 2015), self-reported *truancy* of the child was assessed using one item ("Did you ever skip school"). Details of these measures (including example items) can be retrieved from Supplement 2, available online.

Outcome measures at age 18

Information was collected on various outcomes at age 18, thereby, relying on self- and parent-reports, and a registry. *Aggression* was assessed using self-report (Coccaro, Berman, & Kavoussi, 1997) and parent-report questionnaires (Achenbach & Rescorla, 2003). *Criminality* was assessed with the same self-report tool which was used at age 15 to assess the frequency of violent and non-violent criminal acts. *Rule-breaking behavior* was assessed by the parent-reported Adult Behavior Checklist (ABCL; Achenbach & Rescorla, 2003). *Consequences of antisocial behavior* was assessed through a self-report questionnaire that taps social consequences (e.g., reprimands) caused by involvement in antisocial behaviour (Coccaro et al., 1997). The self-report Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, De La Fuente, & Grant, 1993) 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 (e.g., math, English) in primary school. The grades were obtained through the National School Registry. Details of these measures can be retrieved from Supplement 2, available online.

Cumulative poor functioning at ages 15 and 18

For each follow-up assessment, a cumulative risk index was computed by summing the times 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 Table 1 and 2. At both follow-ups, disruptive behavior subsamples had significantly higher prevalences of maternal mental disorder (follow-up 15 years: 6.4% vs. 5.0%; χ^2 (1, N = 6319) = 5.43, p < .05; 18 years: 5.4% vs. 4.5%; χ^2 (1, N = 3068) = 4.25, p < .05) and paternal mental disorder, (follow-up 15 years: 5.7% vs. 4.0%; χ^2 (1, N = 6319) = 10.20, p < .01; 18 years: 6.2% vs. 3.7%; χ^2 (1, N = 3068) = 4.75, p < .05) compared to non-disruptive behavior samples (follow-up 15 years: n = 4104; 18 years: n = 1878).

Statistical analyses

The continuous outcome variables were substantially skewed, even after data normalization transformations. Therefore, consistent with a large body of research (Côté, Tremblay, Nagin, Zoccolillo, & Vitaro, 2002; Kerr, Tremblay, Pagani, & Vitaro, 1997), dichotomized outcome variables were used. Specifically, echoing prior work (Bechtold, Hipwell, Lewis, Loeber, & Pardini, 2016; Kerr et al., 1997), all outcome measures, except self-reported crime, were dichotomized into high (i.e., the 30% highest scores, 1, which is indicative of low functioning) versus low (i.e., 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 score the dichotomization cutpoints correspond. In line with prior research on the prediction of criminal outcomes (Camp, Skeem, Barchard, Lilienfeld, & Poythress, 2013; Colins, Andershed, & Pardini, 2015), we used dichotomized variables (0 offenses vs. $1 \leq$ offenses) to define future violent and non-violent criminality.

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 = 6319)		Disruptive behavior subsample (n = 2215)	
		Mean (SD)	Range	Mean (SD)	Range
Predictors at 9 years	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
	Parental education level (PR)	4.80 (1.00)	2-6	4.13 (1.00)	2-6
Covariates at 9 years	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 gender male (PR) [n(%)]	2885 (45.7%)	0-1	1120 (50.6%)	0-1
	Nonviolent crime (SR)	0.57 (1.86)	0-52	0.70 (1.94)	0-37
Outcomes at 15 years	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

Table 1 Continued.

Variable function and child age at assessment	Variable	Total sample (N = 6319)		Disruptive behavior subsample (n = 2215)	
		Mean (SD)	Range	Mean (SD)	Range
	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.

Table 2 Descriptive Statistics for Children with Complete Data at Baseline and Age 18 Years

Variable function and child age at assessment	Variable	Mean (SD)	Range	Mean (SD)	Range
Predictors at 9 years					
	Disruptive behavior (PR)	0.72 (1.43)	0-13	1.85 (1.79)	0.5-13
	Maternal mental disorder (Reg.) [n(%)] ^a	159 (5.2%)	0-1	64 (5.4%)	0-1
	Paternal mental disorder (Reg.) [n(%)] ^a	134 (4.4%)	0-1	74 (6.2%)	0-1
Covariates at 9 years					
	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(%)]	1331 (43.4%)	0-1	584 (49.1%)	0-1
Outcomes at 18 years					
	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

Table 2 Continued.

Variable function and child age at assessment	Variable	Total sample (N = 3068)		Disruptive behavior sample (n = 1190)	
		Mean (SD)	Range	Mean (SD)	Range
	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	1023 (33.3)		527 (44.3)	

Note. PR = parent-reported; Reg. = registry; SR = self-reported.

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 (i.e., twins nested within families) is needed in this study to correct for dependency of observations (i.e., 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, like under- and overdispersion. Wald Chi-square tests were used to test the fixed effects. For the fixed effects corresponding odds ratio's (*ORs*) and 95% confidence intervals (*CIs*) were computed and reported.

Poisson or negative binomial models can accommodate non-normality 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 regressions analyses and found that the pattern of the findings were 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 (Evans, 2004), maternal age at childbirth (Chang et al., 2014), paternal age at childbirth (Janecka et al.), and gender of the child (Zahn-Waxler, Shirtcliff, & Marceau, 2008). 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 if parental MD is a risk factor of future maladjustment among children who already display DB, both models (being referred to as Models 3 and 4, respectively) were repeated in a subsample of children who displayed at least some DB (i.e., a raw DB score of 0.5 or higher). Of note, GLMMs were conducted separately for individuals with outcome data at age 15 years ($N = 6319$; DB sample $n = 2215$, 35.1% of total sample at age 15) and for individuals with outcome data at age 18 years ($N = 3068$; elevated DB sample $n = 1190$, 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 ($n = 1696$) and only 126 of these children had a parent with a MD. Therefore, it was not tenable to run the GLMMs. Second, different outcome measures were used across the follow-up assessments, limiting the possibility to test 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 (i.e., 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, there were 1680 out of 7999 children who were not included in the analyses due to 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% vs. 60.2%, $p < .001$) and had parents with lower levels of education ($p < .001$). At age 18 years 3305 out of 6373 children were excluded due to missing data. Significant differences emerged between children with and without missing data in terms of age of the mother at birth ($M = 30.4$, $SD = 4.72$ vs. 30.7 years, $SD = 4.56$, $p = .023$, $d = 0.07$), percentage of boys (48.3% vs. 60.2%, $p < .001$), and parental education level ($p < .001$), but not in terms of paternal age at childbirth.

RESULTS

Outcomes at age 15

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, though the prospective association with reactive aggression was no longer statistically significant. Maternal MD remained positively associated to 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, while maternal MD was positively associated to 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

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, being parent-reported rule-breaking behavior. These prospective relations between paternal MD and future outcomes remained significant in the adjusted models (Model 2), though 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” (i.e., Is there an internalizing or externalizing MD present in the parents?) might be more important than the “who-question” (i.e., Does the mother or the father have a MD?), especially since 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-question” are equally important.

Table 3 Odds ratios and 95% Confidence Intervals of the Fixed Part of the Crude and Adjusted Main Effects Models at Follow-up 15 Years

Predictor	Model	Criminality			Aggression			Problems					Low prosocial (PR)	Cumulative risk ^a
		Violent (SR)	Nonviolent (SR)	OR (95%CI)	Proactive (SR)	Reactive (SR)	Conduct (PR)	Emotional (PR)	Peer (PR)	Alcohol (SR)	Truancy (SR)			
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 = 6319. Model 1 = crude model including the control variables: gender 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 model 1. CI = confidence interval; DB = disruptive behavior; MD = mental disorder; OR = odds ratio; PR = parent-reported; SR = self-reported.

^a Cumulative risk was predicted through negative binomial regressions.

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

Table 4 Odds ratios and 95% Confidence Intervals of the Fixed Part of the Crude and Adjusted Main Effects Models at Follow-up at 18 Years

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

Note. N = 3068. Model 1 = crude model including the control variables: gender 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 model 1. CI = confidence interval; Conseq. = consequences; DB = disruptive behavior; MD = mental disorder; OR = odd ratio; PR = parent-reported; Reg. = registry; Rule-break. = rule-breaking; SR = self-reported.

^a Cumulative risk was predicted through negative binomial regressions.

** $p < 0.01$; * $p < 0.05$.

Table 5 Odds ratios and 95% Confidence Intervals of the Fixed Part of the Crude and Adjusted Main Effects Models at Follow-up 18 years in a Subsample of Children with Disruptive Behavior

Criminality				Aggression			Problems					Cumulative risk ^a	
Violent (SR)		Nonviolent (SR)	Aggression (SR)	Aggression (PR)	Conseq. of antisocial behavior (SR)	Rule-break. (PR)	Emotional (PR)	Alcohol (SR)	Truancy (SR)	Poor school performance (Reg.)			
Predictor	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)	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 = 1190. Model 1 = crude model including the control variables: gender 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 model 1. CI = confidence interval; Conseq. = consequences; MD = mental disorder; OR = odds ratio; PR = parent-reported; Reg. = registry; Rule-break. = rule-breaking; SR = self-reported.

^a Cumulative risk was predicted through negative binomial regressions.

** $p < 0.01$ * $p < 0.05$.

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 a 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 (Breux et al., 2014). 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, while mothers are more focused on social-affective behaviour (Feldman, 2012). 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 (e.g., school suspensions) of antisocial behaviour, 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 a risk factor for reoffending (Piquero, Jennings, & Barnes, 2012), while truancy in adolescence is also a risk factor for later crime (Loeber & Farrington, 2000), mental health problems (Dembo et al., 2012), and academic underachievement (Bridgeland, DiIulio Jr, & Morison, 2006). In addition, poor school performance in adolescence increases the risk for later health problems (Lleras-Muney, 2005), reliance on government assistance, illicit substance use, arrest, and being fired (Lansford, Dodge, Pettit, & Bates). Therefore, paternal MD may not only jeopardize the transition from childhood to adolescence (this study), but also a successful 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 that most clearly affect society as a whole, being 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 behaviour, 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 of first-time offending are less useful to predict reoffending (Cottle, Lee, & Heilbrun, 2001). 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 at age 18. Possibly, children at age 18 become increasingly independent from 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, see Tables 1 and 2), and, therefore, also the likelihood to find 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 a MD. Crucially, fathers have been understudied compared to mothers in studies linking parental MD and child maladjustment (Breaux et al., 2014). 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 the light of evidence that maternal MD is a risk factor for a variety of poor psychosocial outcomes in offspring (S. H. Goodman et al., 2011). 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, while maternal MD has a larger impact on younger children (Connell & Goodman, 2002). Also, prior work suggests that paternal MDs are more strongly associated with child behavioral problems than child internalizing problems (Narayanan & Nærde, 2016; Trautmann-Villalba, Gschwendt, Schmidt, & Laucht, 2006). This is consistent with our results showing that paternal MD in the total sample was associated with increased rates of antisocial behavior (e.g., aggression, crime, and truancy) but not once to emotional problems. Furthermore, our findings are also consistent with evidence that fathers have a larger effect on the development of delinquency in their offspring than mothers (Hoeve et al., 2009), 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 (Fergusson et al., 2005; Wertz et al., 2018) 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 (e.g., conduct problems, emotional problems and low prosocial behaviour) at both follow-up intervals but also of negative outcomes that were based on youth self-report (e.g., proactive aggression, violent criminality, and alcohol use) and information from registries (i.e., 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 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 does not display severe DB and does not meet criteria to warrant a formal ODD or CD diagnosis. Future research is warranted to see if our findings can be replicated in children with severe DB or with formal disruptive behaviour 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 which were assessed at two different follow-ups (Anckarsäter et al., 2011). Evidently, 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 to find significant main effects of parental MDs in children with DB, especially since there is some evidence that the timing of exposure of parental MD matters when studying outcomes in children (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005). Second, specific mental disorders in parents have been associated with different child outcomes (Connell & Goodman, 2002; Ramchandani & Psychogiou), and there is some evidence to suggest that relations between specific parental mental disorders with future child outcomes is influenced by the gender of the parent (Breux et al., 2014). 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 to test the effect of specific MDs (e.g., substance use disorder, major depressive disorder) in parents in general, or in mothers and fathers separately.

Also, regardless of the large number of children and parents included in this study, the number of children with DB who had parents with a MD was rather low. Thus, prevalence issues may also have hampered the likelihood to find 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 a 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 Methods 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 (Farrington & Loeber, 2000), and enhances comparison with prior work that used distribution-based cut-offs (Bechtold et al., 2016; Côté et al., 2002; Kerr et al., 1997). Fourth, officially recorded parental mental disorders are most likely an underestimation of the true extent of parental mental disorders, suggesting that future research also need 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 that are already in treatment for DB. Treatment-as-usual of the child could be augmented with additional screening and, if necessary, treatment of mental health problems in its parents improvement of parental mental health has been associated with better child outcomes (Wesseldijk et al., 2018). Furthermore, the current study clearly underscores the urgency to screen for early-onset child DB in the community, since an accurate identification of these children may eventually increase the likelihood that intervention programs might mitigate or even prevent a developmental pathway towards disruptive behavior disorders and maladjustment in adolescence and adulthood (Kyranides, Fanti, Katsimicha, & Georgiou, 2018).

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 of poor psychosocial functioning in middle and late adolescence.

