Effects of postnatal parental smoking on parent and teacher ratings of ADHD and oppositional symptoms.

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Abstract:

To assess the effects of postnatal parental smoking on subsequent parent and teacher ratings of DSM-IV attention deficit hyperactivity disorder (ADHD) symptoms and oppositional behaviors in children diagnosed with ADHD and their siblings. Children between 5 and 12 years of age with ADHD and their siblings were included. DSM-IV ADHD symptom subscales (Inattentive and hyperactive-impulsive), and oppositionality subscale scores from Conners' Rating Scales were predicted on the basis of parental smoking status in the first 7 years after birth using Generalized Estimating Equations controlling for a range of relevant covariates. Postnatal parental smoking was associated with both parent and teacher ratings of ADHD symptoms and oppositional behavior. After controlling for a number of covariates, several of these relationships were still significant. The risk of maternal smoking for the development of ADHD symptoms does not end during pregnancy. Research on the mechanisms underlying the observed associations is needed.

Keywords: postnatal parenting | parent smoking | ADHD | oppositional behaviors | child development | maternal smoking effects

Article:

Attention deficit hyperactivity disorder (ADHD) is one of the most common psychiatric disorders of childhood, with prevalence estimates ranging from 4% to 8% across cultures (Polanczyk et al., 2007; Visser et al., 2007). ADHD is associated with significantly lower quality of life and a wide range of long term adverse outcomes, including criminality, lower occupational status, substance abuse, lower academic achievement, more driving accidents, and a greater incidence of divorce (Able et al., 2007; Barkley and Cox, 2007; Escobar et al., 2005;

Hakkaart-van Roijen et al., 2007; Klassen et al., 2004; Mannuzza et al., 1991; Mannuzza et al., 1997; Mannuzza et al., 1993, 1998; Mannuzza et al., 1991). Moreover, the public health impact of the disorder and its burden to the health care system and society are significant (Pelham et al., 2007; Ray et al., 2006).

Considerable effort has gone into the identification of etiological factors that lead to the development of ADHD. Evidence from family, adoption, twin, and molecular genetic studies highlights the centrality of genetic factors in the development of ADHD (Faraone and Khan, 2006; Faraone et al., 2005; Waldman and Gizer, 2006). For example, twin studies consistently report heritability estimates of ADHD to be 0.6 to 0.9, which are among the highest estimates for any psychiatric disorder (Faraone et al., 2005; Waldman and Gizer, 2006). Despite the clear role of genetics in accounting for ADHD, a number of environmental factors have also been associated with development of ADHD and other related behavioral problems, including maternal psychopathology (Romano et al., 2006; Shaw et al., 2005), negative parenting styles (Morrell and Murray, 2003), and lead and other metal exposure (Minder et al., 1994; Tuthill, 1996).

The most widely studied nongenetic factor in the development of ADHD, however, has been maternal prenatal smoking, with recent data suggesting that children of mothers who smoke during pregnancy are more than twice as likely to develop ADHD compared with children of mothers who do not smoke (Langley et al., 2005). Studies have shown that individuals whose mothers smoked during pregnancy are more likely to exhibit ADHD-related behaviors measured both categorically (i.e., via diagnostic interviews) or dimensionally (i.e., via parental rating scales), even when controlling for a wide range of potential confounder variables, such as maternal ADHD symptoms and/or other psychiatric disorders (Huizink and Mulder, 2006; Linnet et al., 2003). Moreover, there appears to be a dose-response relationship in that there is a positive correlation between the severity of the behavior problems or risk for the disorder and the amount of smoking reported during pregnancy (Huizink and Mulder, 2006; Linnet et al., 2003). Maternal prenatal smoking has also been shown to affect a range of outcomes in offspring that are related to ADHD, including neuropsychological and cognitive functioning and other disruptive behavior problems (Huizink and Mulder, 2006; Linnet et al., 2003). Several more recent studies have also found significant interactions between genotype (specifically variants in the DAT1, DRD4, and CHRNA4) and prenatal smoking in predicting ADHD-related outcomes (Becker et al., 2008; Neuman et al., 2007; Todd and Neuman, 2007).

Comparatively little work has been done to evaluate the effects of postnatal maternal smoking on subsequent behavior problems in children. The mechanisms underlying increased risk for ADHD in offspring of smoking mothers are likely related to the well-established teratogenic effects of

prenatal nicotine exposure (Paz et al., 2007; Vaglenova et al., 2004). However, it has also been demonstrated that peri- and postnatal exposure to nicotine results in significant changes in brain regions relevant for ADHD and other disruptive behaviors (Britton et al., 2007; Miao et al., 1998). Because children exposed to second-hand smoke after birth receive significant levels of nicotine as measured in saliva (Jarvis et al., 1985; Strachan et al., 1989), it stands to reason that investigating the effects of postnatal smoke exposure on ADHD symptoms would be an important area of research. The few studies that have systematically assessed the independent effects of postnatal maternal smoking on subsequent child behavior problems report inconsistent results. For example, one study reported that postnatal exposure to maternal smoking significantly increased parent ratings of disruptive behavior (including ratings of ADHD symptoms) compared with children who were not so exposed. These effects, however, were rendered insignificant when the effect of prenatal maternal smoking was controlled (Fergusson et al., 1993). By contrast, another study found that persistent maternal smoking (i.e., smoking both before and after pregnancy) was significantly associated with parent-rated behavior problems, whereas prenatal smoking only was not significantly predictive of behavior problems when postnatal smoking was controlled (Maughan et al., 2001). Finally, several studies have reported that both pre- and postnatal maternal smoking independently predict subsequent parent-rated behavior problems in a dose-dependent manner (Weitzman et al., 1992; Williams et al., 1998).

Studies that have investigated the effects of postnatal maternal smoking on subsequent behavior problems in offspring have assessed a range of disruptive behaviors using several different behavioral rating scales. None, however, have explicitly evaluated the DSM-IV symptoms of ADHD. Given the strong findings on the effects of prenatal maternal smoking on ADHD symptoms and diagnosis, we sought to extend this work by examining the effects of postnatal maternal smoking specifically on ADHD symptoms. The purpose of the present study, therefore, was to evaluate the effects of postnatal maternal smoking on ADHD symptoms and other disruptive behaviors in a sample of children ascertained on the basis of an ADHD diagnosis and their siblings, while controlling for a range of important covariates, including prenatal smoking and self-reported maternal ADHD symptoms. We also sought to analyze the effects of postnatal paternal smoking on ADHD outcomes. Primary outcome measures included parent and teacher ratings of DSM-IV ADHD symptoms divided into Inattentive (IA) and hyperactive-impulsive (HI) domains and parent and teacher ratings of oppositional behavior. Consistent with previous studies that have evaluated other related disruptive behaviors, we hypothesized that postnatal smoke exposure (from either mothers or fathers) would be associated with higher ratings of ADHD and oppositional defiant disorder (ODD) symptoms.

METHODS

Participants

TABLE 1. Demographic Characteristics of Sample

	N	Mean (*SD*)/%	Range
ADHD Status (% diagnosed with ADHD)	244	78.7	—
CPRS			
log Opp	244	4.12 (0.24)	3.67–4.59
IA	244	63.00 (14.21)	41–100
HI	244	71.54 (16.36)	40–133
CTRS			
Opp	197	59.22 (18.18)	45–131
IA	197	64.13 (13.36)	41–99
log HI	196	4.09 (0.23)	3.74–4.70
Mother's CAARS T-Score	238	53.85 (13.76)	27–89
Full scale IQ estimate	213	106.74 (14.79)	76–146
Age at CPRS completion	244	8.62 (2.83)	2–17
Race (% white)		79.47	
Gender (% male)		59.84	
ODD/CD comorbidity		35.54	
Maternal smoking status			
Prenatal (% Yes)		7.79[a]	
Postnatal (% Yes)		15.16[a]	
Paternal smoking status			
Postnatal (% Yes)		23.31[a]	

[a]The denominator for these percentages was total number of children (N = 244), rather than total number of families (N = 151) since some mothers reported different smoking behavior for different children.

Opp indicates Oppositionality Subscale; IA, DSM-IV Inattention Subscale.

Participants for the study were 244 children and adolescents drawn from 151 families, of whom 120 were white, 29 were African American, and 2 families were of Asian ethnicity. All analyses controlled for nonindependence of individuals from the same family. For all families in this analysis, parental report (including both rating scales and smoking behavior) was provided by the mother. Demographic information on the sample is shown in Table 1. The families were recruited at 2 academic sites (Duke University Medical Center and University of North Carolina, Greensboro) which are both located in central North Carolina. Participants for the study were drawn from a family-based study to identify genetic factors contributing to ADHD. Families were ascertained through a proband between 5 and 12 years of age, who either had a confirmed diagnosis of ADHD through one of our ADHD specialty clinics or who had a presumed ADHD diagnosis that was subsequently confirmed via a research evaluation. Data regarding ADHD and ODD symptoms were collected for probands (N = 151) and all available siblings (N = 93). It is important to emphasize that although individuals for this analysis were recruited for a genetics study, data from probands and siblings were combined as a single sample and family relations were controlled in the statistical analyses. As such, we report characteristics of the total sample when necessary, rather than probands and siblings separately. The primary reason for this

approach was to look at symptomatology in a continuous manner rather than dichotomizing into diagnostic categories.

Measures

Diagnostic Status

All children in the family, regardless of ADHD status, were given the same assessment battery which included parent diagnostic interviews (NIMH Diagnostic Interview Schedule for Children)(Shaffer et al., 2000), parent and teacher rating scales to assess symptoms in 2 settings (Behavioral Assessment Scale for children [Reynolds and Kamphaus, 1993]; Conners' Rating Scales [Conners, 1997]), as well as IQ screening (Weschler Intelligence Scale for children, Fourth Edition (Wechsler Intelligence Scale for Children, 2003). Adults were also given a limited ADHD assessment which included the Conners' Adult ADHD Rating Scale (Conners et al., 1999). Families were excluded in cases where the child's ADHD diagnosis was not the primary diagnosis (i.e., other significant psychiatric disorders, neurological abnormalities, prematurity) as determined by the study's clinical team, comprised of 3 clinical psychologists (A.D.A., E.M.K., S.K.) and a developmental pediatrician (A.M.L.). Children who were home-schooled were also excluded as this would necessitate the same informant completing the parent and teacher ratings. All data were obtained under IRB-approved procedures at the respective clinical sites, which included written informed consent from parents and verbal assent from the children. By definition, probands from each of the 151 families met criteria for ADHD. Of the 93 siblings that were included in the analysis, 41 (44.1%) met research criteria for ADHD.

Oppositional Behavior and ADHD Symptoms

For purposes of analyses, oppositional behavior and ADHD symptoms were assessed using the Conners' Parent Rating Scale (CPRS) and the Conners Teacher Rating Scale (CTRS). These scales have been used for over 30 years to assess ADHD and related behavior in children and possess excellent psychometric properties (Conners, 1997; Conners et al., 1998a, 1998b). Items from the scale are each rated on a 0 to 3 scale with the following anchors: 0 = "Not at all true (Never, Seldom)," 1 = "Just a little true (Occasionally)," 2 = "Pretty much true (Often, Quite a bit), and 3 = "Very much true (Very Often, Very Frequent). For oppositional behavior, T-scores

(which are age and gender controlled) from the 10-item (CPRS) and 6-item (CTRS) Oppositional subscale were used. For ADHD behaviors, T-scores from the 9-item DSM-IV Inattentive Symptom Subscale and the 9-item DSM-IV HI Symptom Subscale were used. It should be emphasized that the families for this study were ascertained on the basis of at least one child with ADHD. As such, the overall rates of ADHD and ODD or Conduct Disorder (CD) inthe sample were 78.7% and 35.5%, respectively (Table 1). With the exception of teacher ratings of oppositional behavior, probands, and affected siblings differed from unaffected siblings on parent and teacher ratings of ADHD and ODD symptoms (all $p < 0.05$). Probands and affected siblings were more likely to be rated highly on oppositional behaviors by teachers (OR = 2.74), though this was not statistically significant ($p = 0.12$).

Parental Smoking Status

A smoking risk factor questionnaire was also administered to the mothers of all participating children to capture information regarding prenatal and postnatal smoking exposure for each child. Mothers were asked if they “ever smoked” (Y/N), if they smoked during the pregnancy (“prenatal smoking,” Y/N) and if they smoked during the first 7 years of the child's life (“postnatal smoking,” Y/N). If mothers responded affirmatively to the questions about prenatal and postnatal smoking, a follow-up question was also asked regarding how much they smoked per day, with response options as follows: 1 or less cigarette per day, 2 to 4 cigarettes per day, 5 to 14 cigarettes per day, 1524 cigarettes per day, 25 to 34 cigarettes per day, 35 to 44 cigarettes per day, and >44 cigarettes per day. In our analyses, we required a minimum amount of smoking to delineate those women who smoked from those who did not. “Smokers” were defined as women who smoked at least 2 cigarettes a day. “Nonsmokers were defined as women who answered “N” to the question “ever smoked” during the pregnancy. Those women who answered “Y” to “ever smoked” but smoked less than 2 cigarettes a day were eliminated from our analyses. Mothers also completed questions about paternal postnatal smoking for each child and the same definitions were imposed for the paternal “smokers” and “nonsmokers.” Since informants for this study were mothers, there was variability with respect to the amount of time fathers spent time with children during the period of interest (first 7 years of the child's life). Indeed some of the fathers reportedly lived outside the home. Since data were not available on how much time fathers spent with children, we did not further segregate fathers for analysis.

Statistical Analysis

As noted, the primary outcomes for these analyses were subscale T-scores from the CPRS and CTRS. The T-scores from the parent completed Oppositional subscale had to be log-transformed to approach normality for analyses. No adequate transformation on the T-Score distributions for the teacher rated Oppositional subscale could be obtained. As a result, this variable was dichotomized into 2 groups (children whose ratings were <65 and children whose scores were >65) and logistic regression was subsequently used. The T-scores for the CTRS DSM-IV HI subscale also had to be log transformed for analysis. The CPRS DSM-IV HI subscale was not transformed prior to analysis. Similarly, the T-scores for the DSM-IV Inattentive subscales did not need transformation for analysis for either informant (parents or teachers).

The primary independent measure was maternal postnatal smoking status as defined above. Based on the pattern of responding to number of cigarettes smoked per day for positive responses, we dichotomized this frequency variable to assess a potential dose-response relationship among the smokers. The 2 groups for this variable were parents who smoked between 2 and 14 cigarettes per day (n = 18 for mothers; n = 24 for fathers) and those who smoked 15 or more cigarettes per day (n = 19 for mothers; n = 29 for fathers). We considered several potential covariates in our analyses based on the extant literature, including mother's Conners' Adult ADHD Rating Scales T-score and prenatal smoking status, gender of the child, race/ethnicity of the child (Caucasian or African-American; Asian families excluded due to small sample size), age of the child at screening, and whether the child also met research criteria for ODD or Conduct Disorder.

These analyses were carried out using Generalized Estimating Equations with the PROC GENMOD procedure in SAS version 9.1. The Generalized Estimating Equations approach controls for familial correlation among siblings from the same family. PROC GENMOD was used to produce both bivariate and covariate-adjusted analyses. The descriptive analyses of the data set (Table 1) were produced with SAS version 9.1 using PROC FREQ and PROC MEANS statements.

RESULTS

TABLE 2. Bivariate Analyses Examining the Effect of Postnatal Maternal Smoking on Parent and Teacher Ratings of the Children's Behavior

	Postnatal Maternal Smoking Status	Mean/OR	Postnatal Paternal Smoking Status	Mean/OR
CPRS				
Log oppositional	No smoke	4.09*	No smoke	4.11
	Smoke	4.25*	Smoke	4.14
DSM-IV IA	No smoke	69.43**	No smoke	69.78
	Smoke	77.51**	Smoke	73.78
DSM-IV HI	No smoke	69.82*	No smoke	70.19***
	Smoke	81.14*	Smoke	76.16***
CTRS				
Oppositional (T-score ≥65)	Smoke vs. No Smoke	2.67****	Smoke vs. No Smoke	3.38**
DSM-IV IA	No smoke	63.45***	No smoke	63.35
	Smoke	67.63***	Smoke	66.40
Log DSM-IV HI	No smoke	4.08	No smoke	4.07****
	Smoke	4.16	Smoke	4.18****

*$p < 0.001$, **$p < 0.01$, ***$p = 0.05$, ****$p < 0.05$.
IA indicates Inattentive.

Unadjusted analyses for postnatal maternal and paternal smoking on parent and teacher rated behaviors are described in Table 2. Maternal smoking within the first 7 years of a child's life was significantly and positively associated with elevated parent-rated scores for oppositional behavior (log T-score = 4.25 vs. 4.09, $p < 0.001$), inattentive ADHD symptoms (T-score = 77.51 vs. 69.43, $p < 0.01$), and hyperactive impulsive ADHD symptoms (T-score = 81.14 vs. 69.82, $p < 0.001$). Unadjusted analyses of postnatal paternal smoking were not significantly associated with parent ratings of oppositional behavior or inattentive ADHD symptoms. A marginal association was identified between postnatal paternal smoking and parent ratings of hyperactive-impulsive ADHD symptoms (T-score = 76.16 vs. 70.19, $p = 0.054$). We also found evidence for a dose-response effect, particularly for paternal smoking. Parent ratings of oppositional and hyperactive-impulsive ADHD behaviors were higher for those children whose fathers were reported to have smoked >14 cigarettes per day versus those reporting less smoking (log T-scores 4.24 vs. 4.03; T-scores 82.00 vs. 69.08, respectively, $p < 0.05$), and there was a trend for this effect on inattentive ADHD symptoms (78.79 vs. 69.21, $p = 0.07$).

Graphic Table 2

With respect to teacher ratings, postnatal maternal smoking significantly predicted greater scores for oppositional behavior (OR = 2.67, CI: 1.67–6.11, $p < 0.05$) and was marginally associated with inattentive ADHD symptoms (67.63 vs. 63.45, $p = 0.05$). Postnatal paternal smoking was

associated with significantly higher levels of oppositional behavior (OR = 3.38, CI: 1.63–7.02, $p < 0.01$), as well as hyperactive-impulsive ADHD symptoms (log T-scores 4.18 vs. 4.07, $p < 0.05$). There was no evidence of a dose-response relationship for either maternal or paternal postnatal smoking on any of the teacher ratings.

TABLE 3. Adjusted Analyses Examining the Effect of Postnatal Maternal Smoking on Parent Ratings of Behavior

Variable	Status	CPRS–Log Oppositional (Mean/Beta)
Mother's postnatal smoking	No smoke	4.13*
	Smoke	4.26*
Mother's prenatal smoking	No smoke	4.16
	Smoke	4.23
Mother's CAARS		0.003*
Age at CPRS exam		0.008
Race	White	4.18
	African-American	4.21
Gender	Female	4.19
	Male	4.21
ODD/CD Comorbidity	No ODD/CD	N/A
	ODD/CD	N/A
		DSM-IV IA (Mean/Beta)
Mother's postnatal smoking	No smoke	73.67
	Smoke	77.71
Mother's prenatal smoking	No smoke	74.60
	Smoke	76.78
Mother's CAARS		0.149**
Age at CPRS exam		0.2038
Race	White	74.30
	African-American	77.08
Gender	Female	78.10*
	Male	73.28*
ODD/CD comorbidity	No ODD/CD	71.33***
	ODD/CD	80.06***
		DSM-IV HI (Mean/Beta)
Mother's postnatal smoking	No smoke	75.41
	Smoke	81.67
Mother's prenatal smoking	No smoke	76.70
	Smoke	80.38
Mother's CAARS		0.1721*
Age at CPRS exam		−0.1095
Race	White	77.03
	African-American	80.04
Gender	Female	78.82
	Male	78.26
ODD/CD comorbidity	No ODD/CD	71.92***
	ODD/CD	85.15***

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.
IA indicates Inattentive.

TABLE 4. Adjusted Analyses Examining the Effect of Postnatal Maternal Smoking on Teacher Ratings of Behavior

Variable	Status	Oppositional (T-Score ≥65) Beta/OR
Mother's postnatal smoking	Smoke vs. no smoke	1.99 (0.55–7.24)
Mother's prenatal smoking	Smoke vs. no smoke	1.46 (0.25–8.43)
Mother's CAARS		0.0177
Age at CPRS exam		−0.0842
Race	White vs. African-American	0.68 (0.28–1.64)
Gender	Male vs. Female	0.75 (0.40–1.41)
ODD/CD comorbidity	ODD/CD vs. no ODD/CD	N/A
		DSM-IV IA (Means/beta)
Mother's postnatal smoking	No smoke	61.82*
	Smoke	72.79*
Mother's prenatal smoking	No smoke	72.92*
	Smoke	61.69*
Mother's CAARS		−0.1554**
Age at CPRS exam		−0.6326
Race	White	62.71*
	African-American	71.90*
Gender	Female	69.60**
	Male	65.00**
ODD/CD comorbidity	No ODD/CD	66.81
	ODD/CD	67.79
		Log DSM-IV HI (Means/beta)
Mother's postnatal smoking	No smoke	4.13
	Smoke	4.23
Mother's prenatal smoking	No smoke	4.19
	Smoke	4.18
Mother's CAARS		−0.0001
Age at CPRS exam		−0.021*
Race	White	4.11*
	African-American	4.25*
Gender	Female	4.20
	Male	4.17
ODD/CD comorbidity	No ODD/CD	4.18
	ODD/CD	4.19

*$p < 0.01$, **$p < 0.05$.
IA indicates Inattentive.

TABLE 5. Adjusted Analyses Examining the Effect of Postnatal Paternal Smoking on Parent Ratings of Behavior

Variable	Status	CPRS–Log Oppositional (Mean/Beta)
Father's postnatal smoking	No smoke	4.21
	Smoke	4.20
Mother's prenatal smoking		
	No smoke	4.10*
	Smoke	4.31*
Mother's CAARS		0.0039**
Age at CPRS exam		0.008
Race	White	4.19
	African-American	4.22
Gender	Female	4.20
	Male	4.21
ODD/CD comorbidity	No ODD/CD	N/A
	ODD/CD	N/A
		DSM-IV IA (Mean/Beta)
Father's postnatal smoking	No smoke	75.43
	Smoke	76.96
Mother's prenatal smoking		
	No smoke	73.29
	Smoke	79.11
Mother's CAARS		0.17**
Age at CPRS exam		0.18
Race	White	75.24
	African-American	77.15
Gender	Female	78.92*
	Male	73.47*
ODD/CD comorbidity	No ODD/CD	71.68***
	ODD/CD	80.71***
		DSM-IV HI (Mean/Beta)
Father's postnatal smoking	No smoke	77.88
	Smoke	81.27
Mother's prenatal smoking		
	No smoke	75.10
	Smoke	84.06
Mother's CAARS		0.1944*
Age at CPRS exam		−0.1442
Race	White	78.35
	African-American	80.80
Gender	Female	79.92
	Male	79.24
ODD/CD comorbidity	No ODD/CD	72.86***
	ODD/CD	86.29***

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.
IA indicates Inattentive.

TABLE 6. Adjusted Analyses Examining the Effect of Postnatal Paternal Smoking on Teacher Ratings of Behavior

Variable	Status	Oppositional (T-Score ≥65) Beta/OR
Father's postnatal smoking	Smoke vs. no smoke	2.59 (1.15-5.80)*
Mother's prenatal smoking	Smoke vs. no smoke	2.75 (0.60-12.65)
Mother's CAARS		0.058
Age at CPRS exam		−0.0955
Race	White vs. African-American	0.64 (0.25-1.62)
Gender	Male vs. female	0.73 (0.38-1.39)
ODD/CD comorbidity	ODD/CD vs. no ODD/CD	N/A
		DSM-IV IA (Means/beta)
Father's postnatal smoking	No smoke	67.13
	Smoke	68.24
Mother's prenatal smoking		
	No smoke	68.25
	Smoke	67.11
Mother's CAARS		−0.1181
Age at CPRS exam		−0.79*
Race	White	63.80*
	African-American	71.56*
Gender	Female	70.11*
	Male	65.24*
ODD/CD comorbidity	No ODD/CD	67.02
	ODD/CD	68.34
		Log DSM-IV HI (Means/Beta)
Father's postnatal smoking	No smoke	4.16
	Smoke	4.24
Mother's prenatal smoking		
	No smoke	4.16
	Smoke	4.24
Mother's CAARS		0.0004
Age at CPRS exam		−0.0236**
Race	White	4.14*
	African-American	4.26*
Gender	Female	4.21
	Male	4.19
ODD/CD comorbidity	No ODD/CD	4.20
	ODD/CD	4.20

*$p < 0.05$, **$p < 0.01$.
IA indicates Inattentive.

The effects of both maternal and paternal smoking during the first 7 years of the child's life were subsequently examined after controlling for a number of factors that could have influenced the outcomes including maternal self-ratings of ADHD behaviors on the Conners' Adult ADHD Rating Scales, child race, child sex, child age, whether the child had a diagnosis of ODD or CD, and for maternal smoking, self-reported prenatal smoking. Results for these adjusted models are shown in Tables 3, 4 (for postnatal maternal smoking effects on parent and teacher ratings, respectively) and Tables 5, 6 (for postnatal paternal smoking effects on parent and teacher ratings, respectively).

After controlling for all covariates, postnatal maternal smoking was still significantly associated with parent ratings of oppositional behavior (4.26 vs. 4.13, $p < 0.05$). Postnatal maternal smoking was no longer significantly associated with either ratings of HI symptoms or Inattentive symptoms. It is worth noting that in the fully adjusted model examining postnatal maternal smoking, an effect of prenatal maternal smoking was observed for teacher ratings of Inattentive behavior that was in the opposite direction (Table 4). That is, teacher ratings of inattentive behavior were higher for children whose mothers reported no smoking during pregnancy versus those children whose mothers reported smoking during pregnancy.

Postnatal paternal smoking was not significantly associated with any of the parent rated outcomes. Even after controlling for all relevant covariates, there were significant dose-response relations between paternal smoking and parent ratings of oppositional and hyperactive-impulsive ADHD symptoms (log T-scores: 4.29 vs. 4.05; T-scores: 87.07 vs. 73.79, respectively, $p < 0.05$). Further, a trend was observed for a dose-response relation between postnatal paternal smoking and parent ratings of inattentive ADHD symptoms (T-scores: 81.50 vs. 73.52, $p = 0.07$).

Postnatal maternal smoking was significantly associated with teacher ratings of Inattentive ADHD symptoms after controlling for all relevant covariates (T-scores: 72.79 vs. 61.82, $p < 0.01$). Postnatal paternal smoking was significantly associated with teacher ratings of oppositional behavior (OR = 2.59, CI: 1.15-5.80, $p < 0.05$), and a trend was observed for teacher ratings of hyperactive impulsive ADHD behaviors (log T-scores 4.24 vs. 4.16, $p = 0.09$). No dose-response relations were observed for either maternal or paternal postnatal smoking for teacher rated behaviors once covariates were controlled.

DISCUSSION

The present study is one of the first to report on the association between postnatal parental smoking and DSM-IV ADHD symptoms and oppositional behaviors in a sample of children with ADHD and their siblings. In the unadjusted, bivariate analyses, we found that both maternal and paternal smoking after birth during the first 7 years of life were associated with higher levels of oppositional, inattentive, and hyperactive-impulsive symptoms, as rated by parents and teachers. Further, there were significant paternal smoking dose-response relationships observed where children of fathers who smoked more were rated as having more oppositional and ADHD behaviors.

Our adjusted models controlled for maternal self-ratings of ADHD behaviors, child age, child ethnicity, child gender, the presence of a research diagnosis of ODD or CD, and for the maternal smoking analyses, maternal smoking during pregnancy. In these analyses, maternal smoking during the first 7 years of a child's life remained significantly associated with higher parent ratings of oppositional behaviors and teacher ratings of inattentive ADHD behaviors. Postnatal paternal smoking was significantly associated with teacher rated oppositional behavior and trends were observed for both parent and teacher ratings of hyperactive-impulsive behavior. Significant dose-response relationships were observed for postnatal paternal smoking and parent ratings of oppositional behavior and hyperactive-impulsive ADHD symptoms, and a trend was observed for parent ratings of inattentive ADHD behavior.

A number of studies have found strong associations between prenatal smoking and subsequent disruptive behavior problems in offspring (DiFranza et al., 2004; Langley et al., 2007). As noted, a recent review examined findings from 13 population-based and 6 case–control studies of the effects of prenatal maternal smoking specifically on ADHD and related behaviors. With only 2 exceptions, all of these studies found significantly increased risk for an ADHD diagnosis and/or related symptoms as a function of maternal smoking during pregnancy, even when a number of covariates were considered (Langley et al., 2005). While a number of studies have reported on the significant association between maternal smoking after birth and disruptive behavior problems in general (Maughan et al., 2001; Weitzman et al., 1992; Williams et al., 1998), the present findings are among the first to examine this association specifically with DSM-IV ADHD symptoms. Moreover, our study is one of the few that have included data on paternal

smoking. Consistent with these previous studies, our adjusted analyses show significant associations between parental smoking and parent and teacher ratings of oppositional behavior, which are most similar to the more general disruptive behaviors assessed previously. In addition, we found that postnatal parental smoking was also associated with teacher ratings of inattentive ADHD symptoms, which is novel.

There are a number of potential mechanisms that may underlie the present findings. It has been demonstrated that children who live with mothers that smoke receive significant levels of nicotine as measured by salivary cotinine (Jarvis et al., 1985; Strachan et al., 1989). In animal models, postnatal nicotine exposure significantly alters neurodevelopment in transmitter systems that may be relevant to ADHD and related disorders (e.g., serotonin, NaCH) (Britton et al., 2007; Miao et al., 1998). As such, it may be that nicotine and smoke exposure postnatally increases behavior problems directly via alterations in brain function.

Of course, the association between parental smoking and parent and teacher rated behavioral difficulties may be the result of some other factor that was not controlled in the analyses. Moreover, genetic factors may account for this finding in that parents who are prone to smoke both during and after pregnancy may be genetically predisposed to have ADHD symptoms themselves. Although we controlled for maternal ratings of ADHD in the adjusted analyses, this does not necessarily account for potential genetic effects that might manifest themselves during their offspring's childhood. Subsequent studies from this and other samples should examine the interaction between genetic factors and smoking status as independent mediators of child behavior.

The present study is not without its limitations. First, the sample size was relatively small and may have prevented us from identifying associations that we would have otherwise observed. Nevertheless, our clinically ascertained sample was comparable in number to other studies that have investigated the association between maternal smoking and child disruptive behavior (Hill et al., 2000; Landgren et al., 1998; Weissman et al., 1999).

Second, there are several important covariates that were not controlled for that could have influenced the findings, most notably birth weight and SES/psychosocial adversity. Both of these factors have been shown to independently predict subsequent disruptive behavior problems (Breslau and Chilcoat, 2000; Breslau et al., 2000; Gortmaker et al., 1990; Kotimaa et al., 2003; McCarton, 1998). Although it is always challenging to control for all possible relevant covariates, future work would be well served to include these other 2 important variables in the analyses. We also considered child IQ as a potential covariate, but this variable was never significantly associated with the outcome variables in our analyses. For that reason, we removed it from analysis.

Third, there was a relatively low proportion of mothers who reported prenatal smoking in our sample, as defined by our criteria (7.79%). This proportion was lower than the rates of prenatal smoking reported in other population-based studies (Fergusson et al., 1993; Weitzman et al., 1992). This raises some questions about the generalizability of our sample and the appropriateness about drawing inferences regarding the effects of prenatal smoking. This limitation is especially noteworthy since we observed the counterintuitive finding that teacher ratings of inattentive behaviors were higher for children whose mothers reported no smoking during pregnancy. There is no apparent explanation for this discrepant finding, although since postnatal maternal smoking was associated with the opposite finding (teacher ratings higher for children whose mothers did smoke), these data support the potential independence of these processes similar to other studies (Weitzman et al., 1992; Williams et al., 1998). The rates of maternal and paternal smoking after pregnancy (15.16% and 23.21%), however, are more comparable to other population estimates of smoking in adults (~21%)(Early release of selected estimates based on data from the 2006 National Health Interview Survey). It is not known how these rates of pre- and postnatal parental smoking might have influenced the findings, but should be considered when generalizing the results of this study. We also imposed a minimum amount of smoking to qualify as a “smoker” and this, also, could have contributed to the lower rates of smoking in our study population.

Fourth, with respect to paternal smoking, there was likely to be considerable variability with respect to how much time smoking fathers spent with children during the period of interest. Although the majority of fathers (56%) reportedly lived in the same home as the child, a substantial minority of the fathers lived outside the home. It is possible that the effects observed for paternal smoking were carried largely by those fathers who lived in the same home as the child. However, without more detailed information about differences between fathers who lived

in the home and those who did not with respect to time spent with the child, these conclusions cannot be confidently drawn.

Finally, and perhaps most importantly, our measure of parental smoking was assessed solely on the basis of self-report, and is therefore crude. Ideally, obtaining some kind of biological measures to document exposure to nicotine/tobacco smoke would have strengthened the findings and allowed us to draw more solid conclusions. Still the retrospective assessment of maternal smoking behavior is consistent with other studies that have investigated these effects and such self-report has been shown to be adequately reliable (Hudmon et al., 2005; Kenkel et al., 2003; Langley et al., 2005). Certainly, these limitations of the primary independent variable need to be considered when interpreting the results. At the very least, however, the results suggest that parental smoking or variables closely related to the retrospective self-report of parental smoking, are strongly associated with oppositional and ADHD behavior in offspring. Future prospective studies that more precisely quantify parental smoking behavior are warranted based on this study.

The present study extends previous work on the effects of parental smoking on subsequent disruptive behavior problems in children. In particular, it is the first study to document an association between postnatal parental smoking and the DSM-IV ADHD symptoms. ADHD as a disorder has a number of interesting associations with smoking and nicotine, and this finding adds to this growing literature. Individuals with ADHD are known to smoke at rates higher than the general population and this association seems to be driven by the symptoms of the disorder, rather than a clinical diagnosis per se (Fuemmeler et al., 2007; Kollins et al., 2005). ADHD symptoms and genotype have also been shown to interact to increase lifetime risk of smoking (McClernon et al., 2008). Prenatal maternal smoking and genotype also interact to confer strong risk for subsequent ADHD (Becker et al., 2008; Neuman et al., 2007; Todd and Neuman, 2007). Finally, it is well known that nicotine exerts beneficial effects in ADHD and non-ADHD individuals on measure of attention, response inhibition and other tasks known to be disrupted in individuals with ADHD (Conners et al., 1996; Levin et al., 2001; Levin et al., 1998; Levin et al., 1996). Determining the mechanisms that underlie the observation from this study that postnatal parental smoking leads to increases in parent and teacher ratings of behavior problems may help to shed further light on this complex relationship between ADHD and nicotine.

From a clinical perspective, these findings highlight the fact that, in addition to being a risk factor for a wide range of other health problems in children (Rushton, 2004), postnatal parental smoking also increases risk for development of ADHD symptoms. Prevention efforts need to target parents' smoking behaviors even after birth, to help reduce the risk for the development of subsequent behavior problems. These efforts might be best targeted toward parents with a family history of ADHD, nicotine dependence or both.

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