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Attention deficit hyperactivity disorder among children exposed to secondhand smoke: A logistic regression analysis of secondary data

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ABSTRACT

Background: A growing body of literature examines the association of postnatal secondhand smoke exposure with attention deficit hyperactivity disorder (ADHD) in children, but the findings are mixed.
Objective: We compare prevalence of ADHD in children aged 4–15 years who were exposed to postnatal secondhand smoke with prevalence in those who were not exposed, and examine the association of postnatal secondhand smoke exposure with ADHD using both reported and cotinine-measured secondhand smoke exposure.
Design and setting: We analyze secondary data from the 1999–2004 U.S. National Health and Nutrition Examination Surveys.
Participants: Analyses using reported secondhand smoke exposure and cotinine-measured exposure included 6283 and 6033 children aged 4–15 respectively, including 419 and 404 children who either had a reported physician diagnosis of ADHD or were taking stimulant medications.
Methods: The association of secondhand smoke exposure with ADHD was examined by two multiple logistic regression models that differ in the secondhand smoke measurement used.
Results: After controlling for maternal smoking during pregnancy, gender, age, race/ethnicity, preschool attendance, health insurance coverage, and exposure to lead, children with reported secondhand smoke exposure at home were more likely to have ADHD (adjusted odds ratio = 1.5, 95% confidence interval: 1.1-2.0) than those who were not exposed. After controlling for these covariates, children with detectable cotinine levels were more likely to have ADHD (adjusted odds ratio = 1.8, 95% confidence interval: 1.3-2.5) than those with non-detectable levels.
Conclusions: Our findings suggest that secondhand smoke exposure in children is strongly associated with ADHD independent of other risk factors and this association is robust using both measurements of secondhand smoke exposure. Further research is needed to understand the mechanism underlying this association. Nurses and other healthcare professionals can play an important role in encouraging parents to quit smoking to reduce children's exposure to secondhand smoke and their risk of ADHD.

What is already known about the topic?

- Children exposed to secondhand smoke experience a number of negative health effects.
- Children whose mothers smoked while pregnant have greater rates of ADHD.

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- Studies of the relationship between postnatal secondhand smoke exposure and ADHD have led to mixed results.

What this paper adds

- After controlling for maternal smoking while pregnant, other environmental exposures, and socioeconomic factors, secondhand smoke is found to have an independent effect on ADHD.
- The findings are consistent whether secondhand smoke exposure is measured by self-report or serum cotinine.
- Children exposed to secondhand smoke have higher prevalence of ADHD than those not exposed at every age between 4 and 15, except age 4-5 using the reported measure, where the prevalence was slightly lower in the exposed group.
- Our findings provide further support for the notion that postnatal secondhand smoke exposure has an adverse effect on the risk of ADHD in children.

1. Introduction

Despite the documented harm of secondhand smoke exposure for children's physical health (California Environmental Protection Agency, 2005; U.S Department of Health and Human Services, 2006), many children still live with smokers and are exposed to secondhand smoke. In the US in 2005-06, 14.6% of children aged 3-11 lived in homes with smokers and 50.9% of them had detectable serum cotinine (>.05 ng/mL), a metabolite of nicotine. Their geometric mean cotinine level was .11 ng/mL (Max et al., 2009). Internationally, it has been estimated that more than half of all children are exposed to secondhand smoke in their homes (International Consultation on Environmental Tobacco Smoke and Child Health, 1999). Findings from the Global Youth Tobacco Surveys, conducted among students in 132 countries in 1999-2005, indicate that 44% of respondents were exposed to secondhand smoke at home, 56% were exposed in public places, and 47% had parents who smoked (The GTSS Collaborative Group, 2006).

Recently, there has been a growing body of literature linking secondhand smoke exposure, also referred to as environmental tobacco smoke exposure, to children's mental health outcomes (Bandiera et al., 2011; Froehlich et al., 2009; Yolton et al., 2005). One such outcome, attention deficit hyperactivity disorder (ADHD), is a behavioral disorder characterized by a pervasive pattern of hyperactivity-impulsivity and/or inattention. ADHD has become an increasingly common diagnosis in children (Boyle et al., 2011; CDC, 2010) and has been found to increase with age at least through the mid-teenage years (Braun et al., 2006; Froehlich et al., 2009; Kabir et al., 2011). Nurses, particularly school nurses and those in outpatient settings, have played an important role in both the identification and management of ADHD in children (Foreman and Morton, 2011; Krause-Parello and Samms, 2010; Thompson and Ni Bhrolchain, 2011).

Most studies linking secondhand smoke exposure to ADHD in children focus on maternal smoking during pregnancy and find consistent evidence supporting a significantly adverse effect (Hermann et al., 2008; Langley et al., 2005; Schmitz et al., 2006). However, studies examining the association of postnatal secondhand smoke exposure with ADHD are less conclusive. In a 2005 report by the California Environmental Protection Agency (EPA) (California Environmental Protection Agency, 2005), an extensive review of the scientific evidence from major U.S. studies led the authors to conclude that "evidence suggesting a link between postnatal environmental tobacco smoke exposure and impaired cognition and behavior was found to be suggestive, although not entirely consistent" (pp. 4-18). Bandiera et al. (2011) reported that postnatal secondhand smoke exposure was positively associated with *symptoms* of ADHD, but not a *diagnosis* of ADHD. Kabir et al. (2011) and Xu et al. (2010) both found that postnatal secondhand smoke exposure was associated with higher rates of ADHD. However, two studies found that postnatal secondhand smoke exposure was not associated with ADHD (Braun et al., 2006; Froehlich et al., 2009). These mixed results might be attributed to the differences in methods of determining ADHD status, measurement of postnatal secondhand smoke exposure, data used, sample size, exclusion criteria for study sample, and confounding factors included in the study. For example, there has been emerging evidence suggesting that other environmental exposures are linked to ADHD, including lead (Braun et al., 2006; Froehlich et al., 2009) and organophosphate pesticides (Bouchard et al., 2010), and prenatal exposure to organochlorines (Sagiv et al., 2010). Prenatal alcohol exposure has additionally been suggested as a risk factor for ADHD (Banerjee et al., 2007; Knopik et al., 2005). Also, postnatal secondhand smoke exposure is found to be collinear with prenatal maternal smoking (Eskenazi and Castorina, 1999). Therefore, it is important to control for prenatal maternal smoking and other environmental exposures in order to assess the independent effect of postnatal secondhand smoke exposure on ADHD.

This study compares the prevalence of ADHD in children who are exposed to secondhand smoke with those who are not exposed, and examines the association of postnatal secondhand smoke exposure with ADHD using a large nationally representative sample of U.S. children. While the data analyzed in this study are from the U.S., the issue is of international concern given the widespread smoking prevalence and exposure of children to secondhand smoke globally. We extend the previous research by focusing on children who were nonsmokers; by analyzing the independent impact of secondhand smoke exposure after controlling for prenatal tobacco exposure, other environmental exposures, and socioeconomic factors; and by using both reported and cotinine-measured secondhand smoke exposure.

2. Methods

This study was certified as exempt by the University of California, San Francisco Committee on Human Research.

2.1. Data source

We analyzed data from the 1999–2004 U.S. National Health and Nutrition Examination Survey (NHANES), the

most recent years for which ADHD was assessed in children. The survey contains a cross-sectional, nationally representative sample of noninstitutionalized civilians of all ages selected based on a complex sampling design (CDC, 2004a). African Americans and Hispanics are oversampled. The NHANES is conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention, and includes both a household survey component completed during a home interview and a physical examination in a mobile examination center (MEC). Household interviews for survey participants under 16 years of age are conducted with a proxy, usually their parent or guardian. During examination in the MEC, blood samples are drawn for serum cotinine analysis for all individuals aged 3 years and older.

2.2. Outcomes measures

We defined ADHD as parent or guardian reporting of their child's diagnosis of ADHD or stimulant medication use. Reporting of ADHD diagnosis was based on the parent or guardian's answer to the question: "Has a doctor or health professional ever told you that your child had attention deficit disorder?" (CDC, 2004b). Stimulant medication use was based on the parent or guardian's answer to the question: "In the past 30 days, has your child used or taken medication for which a prescription is needed?". Those who answered "yes" were asked to show the interviewer the medication containers of all prescription drugs in order to record the name of the product. Stimulant medications are those with National Drug Codes 03700, 17900, 39500, and 82000, which correspond to amphetamine aspartate/amphetamine sulfate/dextroamphetamine aspartate/dextroamphetamine sulfate, dextroamphetamine sulfate, methylphenidate hydrochloride, and unspecified ADHD medication, respectively (CDC, 2005a,b). These medications are used to treat ADHD (Braun et al., 2006; NIMH, 2011). Because the decision of whether or not to medicate a child is a personal one, and some parents choose not to do so even if recommended by their child's physician (Coletti et al., 2012), we included all children who met the reported diagnosis or medication criteria.

2.3. Measurement of secondhand smoke exposure

Secondhand smoke exposure was measured by two different binary variables (yes or no): reported home exposure and cotinine-measured exposure. A child was considered to have reported home exposure if the household respondent answered yes to the question, "Does anyone who lives here smoke cigarettes, cigars, or pipes anywhere inside this home?" (CDC, 2004a). Serum cotinine is assessed in the NHANES using an isotope dilution, liquid chromatography tandem mass spectrometry method (Bernert et al., 1997; Pirkle et al., 1996). In the 1999-2000 survey cycle, serum cotinine levels as low as 0.05 ng/mL could be detected. Beginning in the middle of the 2001-2002 survey cycle, a more sensitive cotinine assay was used that allowed for a lower detectable limit of 0.015 ng/mL. We used the detectable limit of 0.05 ng/mL for all years for consistency. A child was considered to have cotinine-measured exposure if he or she had a detectable serum cotinine level of 0.05 ng/mL or greater.

2.4. Covariates

Based on the literature review, a number of covariates were considered in this study: prenatal maternal smoking, socioeconomic characteristics (gender, age, race/ethnicity, parental education, and poverty status), preschool attendance (Braun et al., 2006; Froehlich et al., 2009), health insurance coverage (yes or no), and lead exposure. Prenatal maternal smoking was assessed by the question, "Did the child's biological mother smoke at any time while she was pregnant with him/her?" Age was measured in two-year age groups. Race/ethnicity was classified as Non-Hispanic White, Non-Hispanic Black, Mexican American, Other Hispanic, and Other Race. Poverty status was measured using the poverty income ratio (U.S. Census Bureau, 2003), the ratio of family income to the family's poverty threshold taking family size into account (Office of Management and Budget, 1978). Health insurance was included to account for the impact of health care access on diagnosis and medication treatment of ADHD. Lead exposure was measured by blood lead concentration using graphite furnace atomic absorption spectrophotometry, with values below the limit of detection (.3 μg/dL) assigned a value of .2 (0.3 divided by $\sqrt{2}$) (Braun et al., 2006). We categorized lead exposure into quintiles. In a sensitivity analysis described below, we also included exposure to organophosphate pesticides as a covariate (Bouchard et al., 2010). It was measured by urinary concentration of dimethyl alkylphosphates (DMAP) metabolites through lyophilization and chemical derivatization, and analysis through isotope-dilution gas chromatography–tandem mass spectrometry.

2.5. Study sample

The study sample consisted of all nonsmoking children aged 4-15 from the 1999–2004 NHANES. We used a lower bound of age 4 because the questions about ADHD were only asked about children age 4 and older, and an upper bound of age 15 because the question about maternal smoking was only asked for children up to age 15. From the total 7919 eligible children, smokers were excluded using two criteria. First, 150 children aged 4-15 with serum cotinine levels > 10 ng/mL, indicative of active smoking (Pirkle et al., 2006), were excluded. Then, 412 children aged 12-15 who reported having smoked cigarettes at least one day in the past 30 days or used any tobacco products in the past five days, were excluded. These smoking questions were not asked of children younger than 12.

2.6. Analyses

All analyses accounted for the complex survey design and were estimated by incorporating the appropriate sampling weights that adjust for unequal probabilities of sample selection, nonresponse, and sample noncoverage to represent children aged 4-15 years in the U.S. Analyses were conducted using SAS version 9.2 (SAS Institute Inc., 2009). Separate ADHD analyses were conducted using for

reported and cotinine-measured secondhand smoke exposure as the predictor. Weighted prevalence of ADHD was calculated for each population subgroup stratified by secondhand smoke exposure status and covariates considered in this study. Differences in the prevalence of ADHD among different population subgroups were evaluated using bivariate analyses by calculating the chi-square statistics from two-way contingency tables (Fleiss, 1981). Multiple logistic regression models were used to assess the association between secondhand smoke exposure and ADHD while controlling for covariates. The covariates included in the multiple regression models were selected based on findings in the literature and the correlations between covariates and ADHD estimated from the bivariate analyses as mentioned above. Lead, for example, was included in the models because it has been found to be significantly associated with ADHD even after controlling for other factors (Braun et al., 2006; Froehlich et al., 2009). Adjusted odds ratios (AOR) and 95% confidence intervals (CI) were estimated. We considered statistical significance as a two-tailed p-value < 0.05.

Two sensitivity analyses were conducted. In the first, organophosphate exposure was added as a covariate in the multiple regression models along with all the other included covariates. This variable was not included in the main analyses because it was assessed only for a random sample of children aged 6 years and older in NHANES (Bouchard et al., 2010), and only one-third of our study sample had a value for this measure. In the second sensitivity analysis, children whose mothers smoked while pregnant were excluded.

3. Results

After excluding children with missing values on the outcome variable, secondhand smoke exposure and the covariates included in the multiple regression models, the final study sample consisted of 6283 children for the analyses using reported secondhand smoke exposure as the predictor, and 6033 children for the analyses using cotinine-measured exposure as the predictor. Among the final study sample of 6033 children, 22.3% (weighted values, 95% CI: 21.1-26.0) of children had reported secondhand smoke exposure, and 58.3% (95% CI: 53.8-62.9) had detectable serum cotinine levels. This includes 31 children with reported exposure only, 2273 with detectable serum cotinine only, 1259 with both measures, and 2470 with no exposure indicated by either measure. Of those with reported exposure, fully 98% had detectable serum cotinine. Of those who did not have reported exposure, 46% nonetheless had detectable serum cotinine. Table 1 presents the prevalence of ADHD by population subgroups. For the analyses based on reported secondhand smoke exposure, 419 children (8.0% of the 6283 children when weighted) had a reported ADHD diagnosis or stimulant medication use – including 240 with a reported diagnosis of ADHD only, 24 children who took medications only, and 155 who met both criteria. For the analyses using cotinine-measured exposure, 404 (8.0% of the 6033 children when weighted) had a parent-reported ADHD diagnosis or stimulant medication use – including 231 with a reported diagnosis of ADHD only, 24 children who took medications only, and 149 who met both criteria.

Children who had reported secondhand smoke exposure at home had higher prevalence of ADHD than children who were not exposed: 11.5% compared to 6.9% ($p < .01$). Defining secondhand smoke exposure using serum cotinine, there was also a significant difference in ADHD prevalence by exposure level: 9.8% of those exposed compared to 5.4% of those not exposed ($p < .01$). ADHD prevalence differed significantly by prenatal maternal smoking, gender, age, race/ethnicity, preschool attendance, and health insurance coverage.

Fig. 1 shows the prevalence of ADHD by age and secondhand smoke exposure status. For all age groups and both secondhand smoke exposure measures, ADHD prevalence for exposed children exceeds that for children who were not exposed except for children aged 4-5 using the reported secondhand smoke measure. For unexposed children, ADHD prevalence generally increases with age until ages 12-13, and then drops. For exposed children, ADHD prevalence increases from ages 4-5 until ages 8-9, and after then it shows a different pattern depending on the secondhand smoke exposure measurement.

Table 2 shows the results from the multiple logistic regression models on the association between secondhand smoke exposure and ADHD after controlling for covariates. In the model using reported secondhand smoke exposure, children with secondhand smoke exposure at home were 1.5 times as likely to have ADHD as those who were not exposed. The adjusted odds ratio of having ADHD was greater at every age than at age 4-5, and was greatest at age 12-13. The odds of having ADHD was significantly greater among children who had attended preschool, had health insurance, and had higher lead exposure in the 4th or 5th quintile (compared to the first quintile). ADHD was less likely among girls and Mexican American children (compared to boys and non-Hispanic White children). In the model using serum cotinine to measure secondhand smoke exposure, children with detectable cotinine levels were more likely to have ADHD (AOR = 1.8) than those with cotinine values below the detection limit. The impact of other predictors on ADHD followed a similar pattern as for the reported secondhand smoke exposure model except that prenatal maternal smoking showed a significant association with ADHD; non-Hispanic Black children were less likely to have ADHD than non-Hispanic White children, and preschool attendance and lead exposure were no longer significant.

3.1. Sensitivity analyses

In the first sensitivity analysis, the two multiple logistic regression models of ADHD were re-run including organophosphate exposure as a covariate along with all the other variables shown in Table 2. This analysis used the smaller subsample of children with nonmissing data on organophosphate exposure (2026 children for the model using reported exposure, and 1952 children for the model using cotinine-measured exposure). Organophosphate exposure was not significant in either of the multiple regression models after controlling for other

Table 1
Prevalence of ADHD by secondhand smoke exposure and covariates among children aged 4–15: US, 1999–2004.

	Analyses based on reported exposure				Analyses based on cotinine measured exposure			
	Unweighted sample size (*n*)	Number with ADHD	% ADHD (95% CI)	*p*-Value*	Unweighted sample size (*n*)	Number with ADHD	% ADHD (95% CI)	*p*-Value*
Total	6283	419	8.0 (7.1, 8.8)		6033	404	8.0 (7.1, 8.8)	
SHS exposure								
No	4941	288	6.9 (6.1, 7.7)	<.01	2501	98	5.4 (4.1, 6.8)	<.01
Yes	1342	131	11.5 (8.7, 14.3)		3532	306	9.8 (8.3, 11.3)	
Prenatal maternal smoking								
No	5389	310	6.9 (6.0, 7.9)	<.01	5174	306	6.9 (5.9, 7.9)	<.01
Yes	894	109	12.6 (9.6, 15.6)		3041	98	12.9 (9.9, 15.9)	
Gender								
Male	3116	320	11.8 (10.3, 13.4)	<.01	2992	306	11.7 (10.2, 13.2)	<.01
Female	3167	99	3.8 (2.8, 4.8)		3041	98	3.9 (2.9, 4.9)	
Age								
4–5 years	845	15	1.6 (0.5, 2.6)	<.01	779	14	1.6 (0.5, 2.7)	<.01
6–7 years	876	37	4.6 (2.9, 6.4)		821	35	4.6 (2.8, 6.4)	
8–9 years	912	75	9.9 (7.0, 12.8)		879	72	10.0 (7.0, 13.0)	
10–11 years	946	81	9.2 (6.3, 12.1)		911	76	9.0 (6.3, 11.8)	
12–13 years	1476	130	12.8 (10.0, 15.6)		1443	128	12.7 (9.9, 15.6)	
14–15 years	1228	81	8.7 (6.0, 11.4)		1200	79	8.5 (5.6, 11.5)	
Race/ethnicity								
Non-Hispanic White	1622	148	9.0 (7.8, 10.2)	0.01	1565	143	9.0 (7.9, 10.2)	0.01
Non-Hispanic Black	2083	172	8.2 (6.8, 9.7)		1980	166	8.3 (6.8, 9.8)	
Mexican American	2161	69	3.2 (2.2, 4.2)		2095	68	3.3 (2.3, 4.3)	
Other Hispanic	265	20	7.7 (4.0, 11.4)		252	17	6.8 (3.3, 10.2)	
Other Race (incl. multi-racial)	152	10	7.8 (0.0, 15.9)		141	10	8.3 (0.0, 16.9)	
Parent education level								
<High school graduate	2053	108	6.8 (4.7, 8.9)	0.29	1982	103	6.5 (4.3, 8.7)	0.18
High school graduate	1479	93	7.4 (5.6, 9.2)		1419	89	7.3 (5.5, 9.1)	
>High school graduate	2669	212	8.6 (7.4, 9.8)		2553	206	8.7 (7.5, 10.0)	
Poverty income ratio								
Poor (0–0.99)	2013	133	9.3 (6.6, 12.1)	0.37	1931	132	9.7 (6.8, 12.5)	0.31
Low-Income (1–1.99)	1632	107	8.5 (6.3, 10.7)		1573	102	8.2 (6.0, 10.4)	
Middle-Income (2–3.99)	1430	103	7.8 (5.8, 9.7)		1369	97	7.8 (5.7, 9.8)	
High-Income (≥4.0)	837	57	6.5 (4.9, 8.1)		804	55	6.5 (4.9, 8.2)	
Preschool attendance								
No	2046	80	5.7 (3.6, 7.7)	0.02	1972	77	5.8 (3.7, 7.9)	0.02
Yes	4237	339	8.8 (7.9, 9.7)		4061	327	8.8 (7.9, 9.7)	
Covered by health insurance								
No	1083	39	5.0 (3.2, 6.8)	<.01	1040	37	4.5 (3.1, 5.9)	<.01
Yes	5200	380	8.4 (7.4, 9.4)		4993	367	8.5 (7.5, 9.5)	
Lead exposure								
1st quintile (ND–0.8 μg/dL)	1434	71	6.6 (5.1, 8.0)	0.56	1391	69	6.6 (5.1, 8.1)	0.57
2nd quintile (0.9–1.1 μg/dL)	1158	73	8.0 (4.9, 11.1)		1120	71	8.1 (4.9, 11.3)	
3rd quintile (1.2–1.5 μg/dL)	1228	86	8.4 (6.3, 10.5)		1172	83	8.1 (6.0, 10.2)	
4th quintile (1.6–2.2 μg/dL)	1176	83	8.6 (6.9, 10.2)		1135	80	8.7 (6.9, 10.4)	
5th quintile (≥2.3 μg/dL)	1287	106	9.2 (6.2, 12.2)		1215	101	9.3 (6.2, 12.5)	
Organophosphate exposure[a]								
Below detection limit (DL)	260	24	8.9 (5.1, 12.7)	0.08	251	28	9.1 (5.3, 13.0)	0.25
1st level (DL–21.80)	440	33	9.2 (5.9, 12.4)		424	37	9.1 (5.5, 12.7)	
2nd level (22.81–56.52)	441	29	6.5 (3.5, 9.4)		425	32	6.7 (3.6, 9.9)	
3rd level (56.53–165.67)	441	34	7.8 (4.9, 10.6)		427	38	7.4 (4.6, 10.3)	
4th level (>165.67)	441	44	13.2 (7.7, 18.9)		425	49	12.2 (6.7, 17.8)	

Notes: Percentages are based on weighted numbers. Sample sizes may not add up to totals due to missing values. Below detection limit is defined as dimethyl phosphate ≤0.41 nmol/L and dimethyl thiophosphate ≤0.28 nmol/L and dimethyl dithiophosphate ≤0.07 nmol/L.

[a] The listed organophosphate pesticide cut-off is based on the sample in the reported home SHS-exposure model. The DMAP cut-off based on the sample in the cotinine-measured SHS exposure model is as below: below detection limit (DL). 1st level (DL–21.23). 2nd level (21.24–56.46). 3rd level (56.47–163.75). 4th level (>163.75).

* *p*-Value from two-tailed bivariate chi-square test.

covariates, whether entered as quintiles or as a continuous variable. Reported secondhand smoke exposure remained significant, though cotinine-measured exposure was just short of being significant ($p = .06$). We conducted chi-square tests to compare postnatal secondhand smoke exposure, prenatal maternal smoking, lead exposure, and sociodemographic and other characteristics for children with and without organophosphate exposure measures, and found no significant differences between the two groups. In the second sensitivity analysis, the two multiple logistic regression models of ADHD were run by including only children whose mothers did not smoke while pregnant. The odds ratios for both reported secondhand smoke exposure and

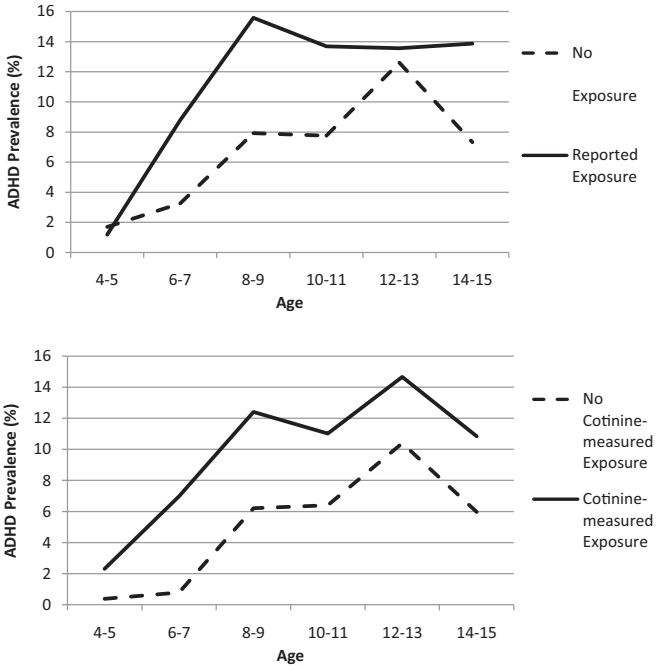

Fig. 1. ADHD prevalence by age and secondhand smoke exposure status: US, 1999-2004. Top panel: secondhand smoke exposure defined as reported by parent. Bottom panel: secondhand smoke exposure defined as detectable serum cotinine.

cotinine-measured exposure remained significant and increased slightly.

4. Discussion

To our knowledge, this study is the first to show a significant adverse association between postnatal secondhand exposure and ADHD regardless of whether using biologically confirmed or reported secondhand smoke exposure measurement in a nationally representative sample of US children. This is particularly striking because our results also show that almost half of the children with no self-reported secondhand smoke exposure do have cotinine-measured exposure. It is now known that many more children than previously thought are exposed to secondhand smoke (Max et al., 2009), and parents may not be aware of all the settings in which their children are exposed. While measuring secondhand smoke exposure using a biomarker such as serum cotinine may give a more realistic assessment of exposure at the time the measurement is taken, cotinine has the disadvantage that it has a very short life (16 h on average), and thus only measures recent exposure (Avila-Tang et al., 2012). Self-report, on the other hand, is likely to reflect exposure over a much longer time period. Thus, each measure has advantages and disadvantages. The consistency in our results using either measure suggests that studies using self-reported or cotinine-measured secondhand smoke exposure can reach the same conclusions.

Four recent studies also used NHANES data to study the association of secondhand smoke exposure with ADHD in children by controlling for selected covariates, but their findings were mixed – two found no significant association (Braun et al., 2006; Froehlich et al., 2009), one found positive association with symptoms of ADHD but no association with a diagnosis of ADHD (Bandiera et al., 2011), and one found a significantly positive association for Mexican American and "other race" children, but no association for non-Hispanic whites or blacks (Xu et al., 2010). Our findings are consistent with the Xu et al. study and the findings of ADHD symptoms from the Bandiera et al. study. We improve on the earlier NHANES studies in several aspects. First, we focused on nonsmoking children by applying two exclusion criteria: excluding children aged 4-15 with serum cotinine level > 10 ng/mL, and excluding children aged 12-15 who either smoked cigarettes in the past 30 days or used any tobacco products in the past five days, while the earlier studies did not exclude smoking children from their study sample (Braun et al., 2006; Xu et al., 2010) or only applied a partial exclusion criterion (Froehlich et al., 2009; Bandiera et al., 2011). Including active smokers in the study sample may bias the estimated association between secondhand smoke exposure and ADHD. Second, we analyzed a larger sample size ($n > 6000$) with a longer study period using the 1999-2004 NHANES data, while the earlier studies had a smaller study sample size (for example, $n < 3000$ in the studies by Froehlich et al., 2009; Bandiera et al., 2011). The smaller sample size might contribute to the lack of detecting statistical significance in the association between secondhand smoke exposure and ADHD. Third, we controlled for organophosphate exposure as an additional environmental exposure, while the earlier studies did not.

Our results are consistent with findings from two recent studies that use other datasets. Kabir et al. (2011) analyzed the 2007 National Survey on Children's Health, and found that U.S. children with parent-reported postnatal secondhand smoke exposure at home were more likely to have

Table 2
Adjusted odds ratios (95% CI) from multiple logistic regression models predicting ADHD among children aged 4–15: US, 1999–2004.

	Adjusted odds ratios ($n = 6283$[a])	Adjusted odds ratios ($n = 6033$[a])
Reported SHS exposure at home		
No	1.0	
Yes	1.5 (1.1, 2.0)*	
Cotinine measured SHS exposure		
No		1.0
Yes		1.8 (1.3, 2.5)*
Prenatal maternal smoking		
No	1.0	1.0
Yes	1.4 (0.9, 2.1)	1.5 (1.0, 2.1)*
Gender		
Male	1.0	1.0
Female	0.3 (0.2, 0.4)*	0.3 (0.2, 0.5)*
Age		
4–5 years	1.0	1.0
6–7 years	2.9 (1.3, 6.5)*	3.0 (1.3, 6.7)*
8–9 years	6.8 (3.4, 13.6)*	7.1 (3.5, 14.1)*
10–11 years	6.5 (3.1, 14.0)*	6.8 (3.0, 15.1)*
12–13 years	9.8 (4.7, 20.4)*	10.2 (4.9, 21.2)*
14–15 years	6.7 (3.0, 15.1)*	6.7 (2.9, 15.8)*
Race/ethnicity		
Non-Hispanic White	1.0	1.0
Non-Hispanic Black	0.8 (0.7, 1.1)	0.8 (0.6, 0.9)*
Mexican American	0.5 (0.3, 0.7)*	0.5 (0.3, 0.7)*
Other Hispanic	0.9 (0.5, 1.8)	0.8 (0.4, 1.6)
Other Race (incl. multi-racial)	0.9 (0.3, 2.8)	1.1 (0.4, 3.0)
Preschool attendance		
No	1.0	1.0
Yes	1.6 (1.1, 2.3)*	1.5 (0.9, 2.3)
Covered by health insurance		
No	1.0	1.0
Yes	1.6 (1.0, 2.4)*	1.8 (1.2, 2.7)*
Lead exposure		
1st quintile (0.2–0.8 μg/dL)	1.0	1.0
2nd quintile (0.9–1.1 μg/dL)	1.2 (0.7, 2.1)	1.1 (0.6, 2.1)
3rd quintile (1.2–1.5 μg/dL)	1.3 (0.8, 2.0)	1.1 (0.7, 1.8)
4th quintile (1.6–2.2 μg/dL)	1.5 (1.1, 2.2)*	1.4 (0.9, 2.1)
5th quintile (≥2.3 μg/dL)	1.6 (1.0, 2.6)*	1.5 (0.9, 2.5)
Nagelkerke R^2[b]	0.136	0.139

[a] Sample size based on children with values on all variables.
[b] Nagelkerke (1991).
* Statistically significant difference from the reference group at p-Value $< .05$, two-tailed test.

ADHD (AOR = 1.44, 95% CI = 1.21–1.72). Using the 2003 Scottish Health Survey, Hamer et al. (2011) found that secondhand smoke exposure measured by salivary cotinine level was significantly associated with hyperactivity among British children. However, neither of these studies controlled for prenatal maternal smoking and environmental exposure to lead and organophosphate pesticides as we did in this study. When we excluded children with prenatal secondhand smoke exposure from the analyses, our results were even stronger, a further indication of the importance of postnatal secondhand smoke exposure as an independent risk factor for ADHD.

We identified children with ADHD based on two criteria: parent-reported ADHD diagnosis or the use of stimulant medications to treat ADHD. Other studies have used only the parent-report diagnosis (Kabir et al., 2011; Xu et al., 2010), both parent report and medication use (Braun et al., 2006), or only parent-reported ADHD symptoms that were assessed by lay interviewers using a standardized diagnostic instrument (Froehlich et al., 2009; Bandiera et al., 2011; Hamer et al., 2011). We used parent report or medication use for several reasons. Because these medications are known to be prescribed to treat ADHD (NIMH, 2011; Braun et al., 2006), it seemed unlikely that a prescription would be given without a diagnosis. In our sample, adding the medication criteria increased our ADHD sample by 3% (24 children). However, requiring that both criteria be met would have reduced our sample by over 60%. Given the relatively small sample size of children with ADHD, we wanted to maintain an adequate sample to analyze the covariates of interest. Furthermore, given the sensitivity surrounding the decision to medicate a child, it is not clear that using both parent report and medication criteria would enable us to do a better job identifying "true" cases. Finally, even with a diagnosis of ADHD, it is possible that some families choose to forgo medications due to the expense. However, it must be acknowledged that physicians may over- or under-diagnose cases of ADHD, and they may over- or under-medicate. Thus, the net impact of using two criteria on the number of ADHD cases identified is not obvious.

Our findings indicate that children exposed to secondhand smoke generally have higher prevalence of ADHD at

every age between 4 and 15, regardless of how secondhand smoke is measured. While there is evidence that ADHD rates drop after the early teens (Braun et al., 2006; Froehlich et al., 2009), it does not mean that the problem resolves itself in time. Our results show that prevalence of ADHD among exposed children is still higher than 10% in older children aged 14-15 years (see Fig. 1). Furthermore, considerable damage can be done to a child's academic achievement and social skills before they reach their mid-teens. Thus, a strong case remains for reducing secondhand smoke exposure among children of all ages.

The inconsistent findings in the literature regarding the association of lead and secondhand smoke exposure and ADHD are curious. Two early studies (Braun et al., 2006; Froehlich et al., 2009) found a significant association. A more recent study (Xu et al., 2010) found lead to be not significant in a bivariate analysis; they did not report the association from multiple analyses. In our study, high levels of lead exposure were found to be significantly associated with ADHD after controlling for secondhand smoke exposure measured by reported exposure and other covariates, but not in the multiple regression model when measured by detectable cotinine. The relationship between lead exposure and ADHD in children should be studied further.

We did not find exposure to organophosphate pesticides to be a significant risk factor for ADHD, contrary to a previous study (Bouchard et al., 2010). This may be due in part to the small sample size of children with organophosphate exposure data who could be analyzed.

Our analyses have some limitations. First, the presence of ADHD is based on parental report of a diagnosis by a doctor or health professional or stimulant medication use. This may lead to an underestimate of cases. A recent study (Froehlich et al., 2007) reported that only 47.9% of the children who met the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria for ADHD actually had a reported diagnosis. Second, mothers may not want to acknowledge smoking during pregnancy, or may not accurately recall their behavior 4-15 years earlier. Thus, our measure of prenatal maternal smoking may not reflect the true exposure. Third, ADHD in adults has been shown to have a genetic component (Stergiakouli and Thapar, 2010), but we were unable to determine whether the children in our sample had a parent with ADHD. Fourth, ADHD has been linked to other prenatal risk factors including exposure to organochlorine (Banerjee et al., 2007), alcohol (Knopik et al., 2005; Sagiv et al., 2010), and smokers other than mother who smoked during pregnancy (Gatzke-Kopp and Beauchaine, 2007; Hsieh et al., 2010); however, we were unable to control for these factors because they are not included in the NHANES data. Finally, because this study is based on cross-sectional data, it is not possible to determine causality.

Our findings that secondhand smoke exposure is a significant risk factor for ADHD in children offers an important reason why parents should be encouraged to quit smoking, and adds to the urgency for better training of health professionals including nurses to help them provide counseling to parents on smoking cessation. Nurses have played a central role in the diagnosis, referral, and management of ADHD for children. Our findings suggest that nurses can play a greater role in the prevention of ADHD, including identifying children at risk of secondhand smoke exposure and educating parents about the potential risk of tobacco smoke exposure for children.

5. Conclusions

Our results indicate that there is a significant adverse association between postnatal secondhand smoke exposure and ADHD in children, even after controlling for prenatal maternal smoking, socioeconomic factors, and lead and pesticide exposure. Further research is warranted to understand the mechanism underlying this association and determine whether it is a result of parenting behavior (Tung et al., 2012), behavioral or neurocognitive effects of secondhand smoke on child development (Yolton et al., 2005), or other factors (Banerjee et al., 2007; Knopik et al., 2005; Sagiv et al., 2010). The relationship of secondhand smoke and ADHD is of growing importance given the large number of children exposed to secondhand smoke worldwide and the growing prevalence of ADHD.

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