

## Secondhand Smoke Exposure and Severity of Attention-Deficit/Hyperactivity Disorder in Preschoolers: A Pilot Investigation

Mini Tandon<sup>1</sup>, Christina N. Lessov-Schlaggar<sup>1</sup>, Rebecca Tillman<sup>1</sup>, Melbourne F. Hovell<sup>2</sup>, Joan Luby<sup>1</sup>

<sup>1</sup>Department of Psychiatry, Washington University School of Medicine, St. Louis, MO, USA

<sup>2</sup>Center for Behavioral Epidemiology and Community Health, San Diego State University, San Diego, CA, USA

\*Corresponding author: [tandonm@psychiatry.wustl.edu](mailto:tandonm@psychiatry.wustl.edu)

### Abstract

**Background:** Less is known about the effects of secondhand smoke (SHS) on mental health as compared with other medical disorders.

**Objective:** The aims of this study were to examine the following: 1) the association of SHS exposure with childhood attention-deficit/hyperactivity (ADHD) and disruptive disorders; and 2) the association of maternal recall of a child's SHS exposure and that child's exposure as measured by bioassay.

**Method:** Sixty children had their saliva collected and assayed for cotinine when they were 4 years old and again when they were 6 years old. Phone interview data were collected to assess maternal recall of the children's exposure to SHS at these ages. The children were assessed annually for ADHD and disruptive disorders. Repeated measures analysis of exposure level by child characteristics was performed.

**Results:** Greater ADHD and conduct disorder severity scores were associated with greater child smoke exposure (ADHD severity,  $P = .043$ ; conduct disorder severity,  $P = .035$ ). A large proportion of mothers reported that their children had no exposure to SHS, despite high levels of measured cotinine in the children's saliva.

**Conclusions:** An association between SHS exposure and ADHD and conduct disorder symptoms was found. Children and parents may benefit from parent education regarding the deleterious effects of SHS.

**Keywords:** secondhand smoke, attention-deficit/hyperactivity disorder, disruptive, preschool, parenting

### Introduction

Approximately 60% of children between the ages of 4 and 11 years old are exposed to secondhand smoke (SHS) in the United States (1). Children are exposed to SHS in multiple locations, most notably at home and in the car (2). In a review of maternal smoking and child behavioral problems, Weitzman and colleagues (3) suggested that, despite study discrepancies, a majority of studies that controlled for confounding factors (e.g., prenatal smoking, maternal education, late prenatal care) found an increased risk for behavior problems among children who had been exposed to SHS. SHS has been associated with an increased risk for conduct disorder and hyperactivity in children (4;5). Limitations of existing SHS studies include the use of parental or self-report of SHS without biomarker

confirmation, which is the most valid measure of exposure (6). In addition, studies often fail to account for parental psychiatric diagnoses that may affect child diagnoses.

Although human studies have been wrought with such challenges, animal studies support the idea that SHS alters normal neurodevelopment in perinatally and postnatally exposed groups of rhesus monkeys (7). Slotkin and colleagues (7) have implicated changes in neurite formation, cell hypertrophy, and cell loss. How such animal findings translate to human neurodevelopment remains unclear. However, such converging evidence from human and animal studies gives rise to the need for further investigation into mental health outcomes among young children who are exposed to SHS. We considered the following hypotheses: 1) increased

SHS is associated with increased severity of attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD), and oppositional defiant disorder (ODD) after controlling for parental ADHD and CD (lifetime); and 2) parents are unaware of and unable to accurately estimate the quantity of SHS exposure that their children received during early childhood. ADHD is a diagnosis ascribed to those who have significant problems with attention and impulsivity in several settings and that is often first diagnosed during early childhood. ODD and CD together are often referred to as *disruptive disorders*. The following study specifically makes use of the definitions found in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV), which was published by the American Psychiatric Association in 1994.

## Methods

### *Study Sample*

Study participants were recruited from community primary care sites for a study that focused on early childhood depression. Participants in this ongoing study had received annual comprehensive diagnostic assessments since they were 3 years old. Maternal reports of child diagnoses of ADHD, CD, and ODD were collected with the use of valid and reliable age-appropriate psychiatric assessments for children between the ages of 3 and 13 years; these included the Preschool Age Psychiatric Assessment (8) and the Child and Adolescent Psychiatric Assessment (9). Mothers reported diagnoses of ADHD and CD (lifetime) for themselves and for the children's fathers via the Family Interview for Genetic Studies (10). The mothers reported on the fathers given the fathers' low participation rates. Parental ODD was not addressed. The original sample was not ascertained for parents with ADHD, for parents with depression, or for parents who smoked. All child participants were asked to provide saliva as part of the original parent study. During the last several years of the study, additional detailed phone interview data were collected to address pregnancy and post-pregnancy smoking as well as maternal recall of the children's exposure to SHS at the ages of 4 and 6 years, when saliva samples were obtained and frozen (November 2003/May 2005 and October 2005/April 2007, respectively). These children were an average age of 11 years and 2 months old (standard deviation, 5 months) when their mothers were contacted by telephone. Saliva samples were assayed for cotinine, which is a valid and reliable biochemical marker of nicotine exposure that has excellent stability when frozen (half life,  $\approx 17$  hours) (11).

## Statistical Analysis

Repeated measures analysis of SHS exposure (according to cotinine levels at the ages of 4 and 6 years) and child characteristics was performed with the use of SAS statistical software version 9.2 (12). Separate univariate repeated measures mixed models with unstructured covariance structures were used to model the effects of potential predictors on SHS exposure levels. Initially, models were run without covariates. Follow-up analyses included maternal reports of paternal ADHD and paternal CD as covariates. The results of these models are presented in the following Results section. Maternal ADHD and CD were not included because no mothers had CD and only one mother had ADHD. The dependent variable was the participant's SHS exposure, which was measured via the cotinine level (continuous). Independent variables in the separate mixed models were the participant's age and gender; SHS exposure (mother's recall [yes/no] of child's exposure to SHS); DSM-IV ADHD diagnosis; DSM-IV ADHD severity score (according to a summary of all 18 DSM-IV symptoms, of 9 inattentive symptoms, or of 9 hyperactive/impulsive symptoms); DSM-IV CD, ODD, and CD/ODD severity scores (according to a summary of all 15 DSM-IV CD symptoms, of 8 DSM-IV ODD symptoms, or of 23 DSM-IV CD and ODD symptoms); family poverty (family income of  $\leq \$20,000$ /year); and mother's self-report of prenatal smoking status (yes/no).

## Results

Cotinine levels were measured in 60 participants. There were 50 participants with saliva available at the age of 4 years and 49 participants with saliva available at the age of 6 years. The mothers of the participants included 13 women who had ever smoked and 11 women who were smokers at the time of the interview. Descriptive statistics for the independent variables in the mixed models are presented in Table 1.

Results of the univariate repeated measures mixed models of cotinine levels are shown in Table 2. These models included only 59 participants, because paternal ADHD and CD data were not available for one participant. Prenatal smoke exposure was associated with early childhood smoke exposure, which was measured as SHS (cotinine level) in this sample (estimate [Est] = 1.62; standard error [SE] = 0.62; F-distribution [F] = 6.89; degrees of freedom [df] = 1, 55;  $P = .011$ ). Child diagnoses of ADHD, CD, and ODD were not associated with SHS. However, higher ADHD and CD severity scores were associated with increased levels of child SHS exposure (*ADHD severity*: Est = 0.11; SE = 0.05; F

= 4.29; *df* = 1, 57; *P* = .043; *CD severity*: *Est* = 0.36; *SE* = 0.17; *F* = 4.66; *df* = 1, 57; *P* = .035). There was also an association between higher levels of SHS exposure and higher levels of poverty (*Est* = 1.29; *SE* = 0.63; *F* = 4.15; *df* = 1, 53; *P* = .047).

**TABLE 1. Characteristics of study participants (N=60) at 4 and 6 years of age\***

Characteristic	Age 4 (N=50)		Age 6 (N=49)	
	%	N	%	N
At or below poverty (≤\$20,000/year)	18.4	9	23.4	11
Male gender	60.0	30	53.1	26
Reported no child SHS exposure	83.3	40	77.1	37
Child ADHD	8.0	4	22.4	11
Child CD and/or ODD	24.0	12	26.5	13
Mother reported prenatal smoking	16.7	8	16.7	8
	Mean	SD	Mean	SD
Child ADHD severity	3.12	3.54	4.00	4.14
Child ADHD inattentive severity	1.48	1.94	2.94	2.70
Child ADHD hyperactive/impulsive severity	1.64	1.89	1.31	2.32
Child CD severity	0.84	1.15	0.92	1.17
Child ODD severity	2.22	2.00	2.14	1.91
Child CD/ODD severity	3.06	2.87	3.00	2.75

*Note:* \*There were 60 children with cotinine levels at age 4, age 6, or both age 4 and age 6.

*ADHD*, Attention-deficit/hyperactivity disorder; *CD*, conduct disorder; *ODD*, oppositional defiant disorder; *SD*, standard deviation; *SHS*, secondhand smoke

A mother’s recall of her child’s exposure to SHS was associated with greater child SHS exposure (*Est* = 1.69; *SE* = 0.35; *F* = 23.84; *df* = 1, 55; *P* < .001). However, even at high levels of child SHS, as indicated by child saliva cotinine levels of more than 0.70 ng/ml, a large proportion of mothers still reported that their children had no exposure to SHS (54.6% at the age of 4 years and 38.5% at the age of 6 years).

**TABLE 2. Univariate repeated measures mixed models of salivary cotinine levels in 59 children\***

Characteristic	Est.	SE	F	df	P value
Age 4 vs. age 6	0.73	0.39	3.49	1, 57	.067
At or below poverty (≤\$20,000/year)	1.29	0.63	4.15	1, 53	.047
Male vs. female	-1.03	0.47	4.83	1, 56	.032
Reported child SHS exposure	1.69	0.35	23.84	1, 55	<.001
Child ADHD	0.82	0.48	3.01	1, 57	.088
Child ADHD severity	0.11	0.05	4.29	1, 57	.043
Child ADHD inattentive severity	0.12	0.08	2.29	1, 57	.136
Child ADHD hyperactive/impulsive severity	0.14	0.09	2.47	1, 57	.121
Child CD and/or ODD	0.69	0.44	2.44	1, 57	.124
Child CD severity	0.36	0.17	4.66	1, 57	.035
Child ODD severity	0.14	0.10	2.05	1, 57	.158
Child CD/ODD severity	0.14	0.07	3.72	1, 57	.059
Mother reported prenatal smoking	1.62	0.62	6.89	1, 55	.011

*Note:* \*Paternal ADHD and CD diagnoses were used as covariants

*ADHD*, Attention-deficit/hyperactivity disorder; *CD*, conduct disorder; *ODD*, oppositional defiant disorder; *SHS*, secondhand smoke; *Est.*, estimate; *SE*, standard error; *df*, degrees of freedom

**Discussion**

Categorical diagnoses of ADHD, CD, and ODD in children were not associated with SHS in this study. However, the severity of child ADHD and CD was associated with SHS, thereby warranting further investigation in larger samples.

These study findings also shed light on a lack of maternal recall of nicotine exposure in children despite biomarker evidence. Whether parents underreported child SHS exposure or were blind to other sources of potential exposure (e.g., a smoker driving the child to school, the presence of contaminated surfaces or clothing) warrants study.

This pilot study faced important limitations, including the small sample size; the inability to assess parents’ personalities, parenting practices, and executive function skills; and the lack of genetic analyses with respect to ADHD and smoking. Despite these limitations, the study results inform the underinvestigated role of SHS exposure in children as it relates to ADHD. If SHS is confirmed as playing a role in ADHD, an additional impetus may be justified for the prevention and control of SHS exposure to prevent ADHD or its exacerbations.

Parental education may be one avenue to use to reduce harm to families, especially to children who are in sensitive developmental stages, such as early childhood. In accordance with Hovell and Hughes’ ecologic model (13), future studies should emphasize the importance of smoking only outside of the home and car and of caretakers protecting their children from SHS even from incidental sources in the community (e.g., visiting a friend who smokes). Larger study samples that control for maternal smoking during pregnancy and maternal education are warranted. These study findings highlight the potential role of SHS in the development of ADHD and disruptive behavior as well as the need for pertinent education.

**Clinical Significance**

The present study investigated the effects of SHS on ADHD and disruptive disorders with the use of both maternal reports and bioassay data (cotinine) for preschool-aged children. The severity of SHS exposure was associated with the severity of ADHD and CD symptomatology after controlling for key covariates. Furthermore, these findings suggest that mothers were either unaware of their children’s exposure to SHS or unable to recall it. In either case, understanding the mechanisms by which SHS influences the severity of psychopathology during childhood seems worthy of investigation. Until such findings can be clarified in larger investigations, it may be helpful to teach parents that no amount of

SHS is considered safe. Such findings, despite study size limitations, have significant public health implications. Larger investigations that detail the associations between SHS exposure during early childhood and neurocognition and behavior are now warranted.

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