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Associations Between Second Hand Smoke Exposure and Sleep Patterns in Children

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Abstract

Objectives—Exposure to second hand smoke has been linked with multiple health problems in childhood. However, there have been limited investigations into the potential effect of second hand smoke exposure on child sleep patterns. The area warrants investigations since inadequate sleep in children has been linked to daytime emotional dysregulation, behavior problems, learning difficulties, and obesity, all of which carry significant morbidity beyond the respiratory ailments traditionally associated with second hand smoke exposure. The objective of this study was to investigate the relationship between exposure to second hand smoke and child sleep patterns among a group of children with asthma who were regularly exposed to tobacco smoke at home.

Methods—We studied 219 children who were enrolled in an asthma intervention trial and regularly exposed to second hand smoke. Serum cotinine was used to measure exposure to tobacco smoke, and sleep patterns were assessed by parent report using the Children's Sleep Habits Questionnaire (CSHQ). Covariates in adjusted analyses included: sex, age, race, maternal marital status, education, and income, prenatal tobacco exposure, maternal depression, HOME total score, household density, asthma severity, and use of asthma medications.

Results—Exposure to second hand smoke was associated with sleep problems including longer sleep onset delay (p=.004), sleep disordered breathing (p = .02), parasomnias (p=.002), daytime sleepiness (p=.022), and overall sleep disturbance (p=.0002).

Conclusions—We conclude that exposure to second hand smoke is associated with increased sleep problems among children with asthma.

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Keywords

passive smoking; environmental exposure; sleep; child; asthma

Introduction

Appropriate sleep quality and quantity are increasingly being recognized as critical elements for many aspects of child health and development. For children, inadequate sleep has been linked with poor school performance,[1] somatic complaints,[1] behavior problems,[2-5] and mental health problems.[1, 6] In addition, sleep problems during childhood are associated with increased incidence of anxiety and depression, aggressive behaviors, and attention problems in adulthood, suggesting a lasting impact of sleep problems on mental health.[7] Poor childhood sleep also predicts the development of obesity and its associated morbidities,[8, 9] indicating an important influence on health outcomes.

Over 25 percent of children experience some type of sleep problem during childhood.[1, 10-12] Among children with asthma, the prevalence of sleep problems is higher, with 40-60% having some difficulty.[13, 14] Children with asthma are nearly four times more likely to experience sleep disordered breathing (SDB),[15] resulting in sleep disruption and decreased sleep efficiency,[16] reduced sleep quality, increased nighttime activity levels,[17] and more difficulties with daytime sleepiness.[16, 18] Sleep efficiency has been shown to improve with effective treatment of asthma symptoms,[16] but even children with clinically stable asthma have worse sleep quality and more daytime sleepiness than children without asthma.[19]

Tobacco exposure is a risk factor for sleep problems in adolescents and adults. Cigarette smoking is associated with changes in sleep architecture, with smokers experiencing longer latency to sleep initiation and lighter sleep.[20] Adult and adolescent smokers report more sleep problems such as trouble initiating sleep, maintaining sleep, waking, and daytime sleepiness.[21-24] Women who smoke during pregnancy are more likely to report insufficient sleep, difficulty initiating sleep, early morning waking, short sleep duration, snoring, and excessive daytime sleepiness when compared with pregnant women who do not smoke. Interestingly, women who are nonsmokers but exposed to second hand smoke (SHS) during pregnancy also report more difficulties with sleep including insufficient sleep, difficulty initiating sleep, and short sleep duration compared with those not exposed.[25]

This effect of SHS smoke on sleep in adult women raises concerns about the potential impact on children whose family members smoke. There is little research on the influence of tobacco smoke exposure on sleep patterns in childhood. Young children who are exposed to tobacco smoke either prenatally or postnatally are reported to have poorer sleep quality[26] and more symptoms of sleep disordered breathing[27] than those who are not exposed. Because tobacco smoke is a known contributor to asthma severity,[28-30] exposure to SHS may have a particularly marked effect on the sleep of children with asthma. Although this possibility has received little empirical investigation, SHS exposure has been associated with increased night wakings in children with asthma.[31] A major limitation of these studies linking SHS and sleep difficulties in children[26, 27, 31] is that they have relied on parental

reports of exposure instead of more precise and objective biological markers of tobacco exposure.

The objective of this study was to examine the relationship between SHS exposure and sleep patterns among a group of children with asthma. We used a biomarker of tobacco exposure, serum cotinine, to objectively quantify exposure, and a validated pediatric sleep survey[32] to characterize sleep patterns. We hypothesized that children with asthma who are exposed to higher levels of SHS would exhibit more sleep problems, as reported by parents, than children with lower levels of exposure.

Patients and Methods

This study utilized the Cincinnati Asthma Prevention (CAP) Study, an asthma intervention trial based on environmental modifications to the home in the form of high-efficiency particulate air cleaners, and outcomes focused on asthma symptoms, health care utilization, and pulmonary function. For the current study, SHS exposure, child sleep patterns, and potential covariates were measured prior to initiation of the asthma intervention.

Recruitment and enrollment procedures of the 6-12 year old children are described in detail elsewhere.[33] Briefly, all children in the sample had doctor diagnosed asthma that was treated within the previous year and exposure to SHS from at least five cigarettes per day at home by parental report. Children were identified by hospital and clinic billing records, and parents of 1678 children were contacted for completion of a screening survey and to request participation if eligible. Children were excluded if they had other respiratory diseases, heart disease, were mentally retarded, or had other serious conditions barring participation in the study. Of the 348 eligible participants, 232 enrolled and completed the main study (67% participation rate), and 219 had complete data pertinent to the current study and were retained for the analysis. Protocols were approved by the institutional review board.

We collected detailed survey data regarding the child's daily exposure to SHS in the home, car, and other locations, including hours of exposure, number of cigarettes per day, whether the child was in the same room during smoking, and if windows were open during car exposure. SHS exposure was also objectively measured using serum cotinine levels detected in samples collected at the baseline home visit and are our primary measure of exposure. Cotinine, a metabolite of nicotine, is a reliable biomarker of exposure to tobacco smoke.[34] Serum levels provide a short-term view of exposure over the previous 48-72 hours. However, due to stability of exposure patterns over time, a one-time cotinine measurement is considered representative of typical daily exposure.[34] Cotinine was measured in serum by the Centers for Disease Control using published methods involving high-performance liquid chromatography linked to atmospheric-pressure chemical ionization tandem mass spectrometry.[35] We applied a log base 2 transformation for analysis of serum cotinine because of the skewed distribution of the raw data. This provides for a simpler interpretation of the coefficients from the regression analyses in which there is an increase in the sleep scale equal to the coefficient for log cotinine for each doubling of the cotinine level.

The Children's Sleep Habits Questionnaire (CSHQ)[3] was used to measure child sleep patterns within the past 2 weeks as reported by the primary caregiver. The CSHQ yields a total sleep disturbance score and scores for eight scales (bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, night wakings, parasomnias, sleep disordered breathing, daytime sleepiness). This instrument is used in clinical and research settings to provide a broad description of child sleep patterns. Internal consistency measures for the entire scale are high ($\alpha = 0.68$ among a community sample, $\alpha = 0.78$ among a clinical sample), and test-retest reliabilities among the scales are high ($r = 0.62$ to 0.79). A total CSHQ score ≥41 has sensitivity and specificity of 0.80 and 0.72, respectively, properly classifying 80% of a group with clinically relevant sleep disorders. To ensure uniform and thorough completion, this measure was administered as an interview in which caregiver responses were recorded by a trained research assistant.

Asthma severity was reported as mild, moderate, severe, or very severe by the child's caregiver. Severe and very severe categories were combined due to small group sizes. Parent report of asthma symptoms is an effective means for characterizing child asthma and is not enhanced by asthma diaries or pulmonary function testing.[36] Parents additionally reported asthma medication use by the child including short-acting bronchodilators, long-acting inhaled steroids, and oral steroids prescribed for acute exacerbations.

Other measured covariates were maternal depression (Beck Depression Inventory, Second Edition),[37] and quality of the home environment, measured with the Home Observation for Measurement of the Environment (HOME) instrument for elementary school aged children.[38] The HOME is primarily an observational tool that assesses the quality of the home environment, including physical characteristics, variety of stimulation, and nurturing behavior from the parent and was completed at a 12-month follow-up visit. For 14 participants, this assessment was missing, and imputed values were used based on age, race, and gender generated by SOLAS™ software.[39]

Univariate analysis involved inspection of frequencies and estimation of means with the associated standard deviation. Due to non-normal distributions, serum cotinine is reported as a geometric mean and 95% confidence interval, and household income is reported as median and 25th and 75th percentiles. We used linear regression for the daytime sleepiness and total sleep disturbance scales. The distributions of the other sleep scales reflected varying degrees of non-normality, so responses were dichotomized, based on approximately the 75th percentile, and logistic regression was used. For each outcome measure, three models were developed to reflect (1) the simple bivariate association between exposure and sleep, (2) the association after adjustment for all covariates (age, sex, race, maternal smoking during pregnancy, marital status, maternal education, household income, household density and number of siblings, maternal depression, HOME score, asthma severity, and asthma medication use), and (3) the association after adjustment for important covariates representing the most statistically parsimonious final model. For the final models, age, sex, and asthma severity were retained irrespective of statistical significance. Other covariates were retained if they accounted for significant variance on the given sleep scale based on $p < .05$. Also, if removal of the covariate from the model was associated with a greater than

10% change in the regression coefficient for serum cotinine, it was retained in the model. SAS version 9.1[40] was used for all analyses.

Results

Descriptive information on the sample is summarized in Table 1. The mean age of the subjects at the baseline visit was 9.4 years. Sixty one percent of the children were boys and 56% were reported to be African American. Children in the sample were exposed to a median of 13 cigarettes each day in their homes as reported by parents, and the geometric mean serum cotinine level for the sample was 1.16 ng/mL. The correlation between serum cotinine and parent-reported exposure was 0.39 ($p < 0.0001$) for the full sample.

Mean values for overall sleep disturbance and sleep scale scores for children in our sample fell between the clinical and control samples reported by Owens.[32] Internal consistency for the sleep measure was also comparable to Owens' findings. Surprisingly, 93% of the children in the sample had a total sleep disturbance score that would be considered clinically relevant on the CSHQ (≥41). The mean sleep time reported by parents was 9.6 hours per night.

In bivariate analyses, the associations between the log of serum cotinine and child sleep patterns were significant for bedtime resistance, sleep anxiety, parasomnias, sleep disordered breathing, daytime sleepiness, and total sleep disturbance, but not for sleep onset delay or sleep duration. (Table 2) There was no association between total duration of nighttime sleep and serum cotinine.

In multivariable analyses including all potential covariates of child sleep patterns, we found that higher levels of SHS exposure were significantly associated with higher scores (i.e., more problems) on the sleep onset delay, parasomnias, daytime sleepiness, and total sleep disturbance scales. Final models, including only those covariates that had a relationship with the sleep scale of interest or impacted the coefficient for cotinine, revealed significant associations between SHS exposure and increases in sleep onset delay, parasomnias, sleep disordered breathing, daytime sleepiness, and overall sleep disturbance. (Table 2)

Several covariates remained in our final models due to associations with sleep scales. Increasing age was significantly associated with decreased bedtime resistance, more problems with sleep duration, less sleep anxiety, and lower overall sleep disturbance. More severe asthma was associated with more problems with sleep duration, and more frequent night wakings. Maternal smoking during pregnancy was associated with decreased sleep onset delay. A non-married parent was associated with fewer parasomnias and increased daytime sleepiness. Lower family income was associated with decreased sleep duration and decreased daytime sleepiness. Higher levels of maternal depression were associated with more frequent parasomnias, increased daytime sleepiness, and greater overall sleep disturbance. More siblings and increased housing density were associated with decreased sleep disordered breathing. Finally, the use of long-term inhaled asthma medications was associated with fewer parasomnias.

We found cotinine by sex interactions for the sleep onset delay (OR = 0.63, p = .011) and sleep anxiety scales (OR = 1.47, p = .05) so we performed a sex-stratified regression analyses on those scales, controlling for the covariates retained in the final models for the full sample. For boys, a statistically significant relationship was found between serum cotinine and higher scores on sleep anxiety (OR = 1.54, p = .003). For girls, a statistically significant relationship was found between serum cotinine and sleep onset delay (OR = 1.54, p = .008).

Discussion

In children with asthma, we found that SHS exposure was associated with greater parent-reported sleep problems. Specifically, as SHS exposure increased, parents reported that their children had longer delays in sleep onset, more frequent parasomnias and sleep disordered breathing, increased daytime sleepiness, and greater overall sleep disturbance. Two sleep scales showed significant sex by cotinine interactions. In regression analyses stratified by sex, higher exposure to SHS was associated with greater sleep anxiety in boys and greater sleep onset delay in girls.

SHS exposure was associated with an increased incidence of parasomnias in this sample of children. Parasomnias reflect a partial arousal from either non-rapid eye movement (NREM) or rapid eye movement (REM) sleep and, while usually benign, can be highly distressing to children and their families. Over 80% of preschoolers experience parasomnias, but their incidence decreases with age.[41] Adult men who smoke report more nightmares and disturbing dreams than men who do not smoke, but there has been no reported association for women.[24]

Boys in our study experienced greater sleep anxiety with increasing SHS exposure. Nighttime fears are reported by up to 79% of 8 to 16 year olds. In contrast with our findings, however, they have been reported more frequently among girls (72%) than boys (55%).[42] For girls in our study, greater SHS exposure was associated with greater sleep onset delay which is consistent with reports that both men and women who smoke cigarettes have increased difficulty initiating sleep.[43] No other studies have investigated the relationship between SHS and sleep onset delays among children.

The exact mechanism by which SHS exposure may impact children's sleep is not clear. We briefly explore three possible explanations – exacerbation of respiratory symptoms, nicotine arousal mechanisms, and symptoms of abstinence. In adults, smoking is known to exacerbate respiratory disorders such as obstructive sleep apnea,[24, 44] and SHS exposure has been associated with increased snoring in pregnant women.[25] Among children, parent-reported maternal smoking is associated with increased snoring,[27] and nighttime respiratory symptoms are exacerbated by exposure to SHS.[31] Indeed, higher levels of SHS exposure were associated with more sleep disordered breathing among children in our study. It is likely that SHS exposure acts as an upper airway irritant, increasing symptoms of sleep disordered breathing and thus contributing to overall sleep disturbance among children with asthma.

While adolescent and adult smokers report disturbances in sleep,[22–24, 43, 45] a clear causal relationship between tobacco and sleep disorders has been difficult to establish.[44] Nicotine is a stimulant that may contribute to increased arousal and attention among smokers likely by stimulating neurotransmission of acetylcholine and triggering activation of the dopaminergic system in the brain.[44, 46] Nicotine is associated with altered sleep architecture resulting in reduced sleep time and efficiency, longer sleep latency, lighter sleep, and reduced REM sleep.[20, 47]

Frequent night wakings among smokers are often attributed to withdrawal during sleep. During the early stages of smoking abstinence, adult smokers experience difficulty falling asleep, reduced sleep efficiency, and longer latency to REM sleep.[48] Colrain reports increased night wakings and sleepiness as the most consistent finding of research on smoking cessation.[49] Insomnia, sleep disturbance, and anxiety are also symptoms of nicotine withdrawal listed in the DSM-IV.[50] These symptoms in many ways resemble those reported in our sample. It is possible that children exposed to SHS experience a degree of nicotine withdrawal during sleep, resulting in disruptions in the normal sleep process. Although withdrawal symptoms among children exposed to SHS have to our knowledge not been noted in the literature, several studies present evidence that newborns who were prenatally exposed to tobacco experience nicotine withdrawal shortly after birth.[51–53] This area requires further study.

SHS exposure results in a much smaller amount of nicotine intake than active smoking. Studies of SHS effects on sleep have included reports of sleep disturbances among adult men,[43] pregnant women[25] and preschool aged children[26] indicating that even small amounts of exposure to nicotine, as would occur in SHS exposure, are sufficient to adversely affect sleep. Our study contributes additional evidence that SHS impacts sleep among school-aged children with asthma.

This study is not without limitations. All children in the study had asthma, so the results may not be generalizable to populations of children without asthma. Still, these results may be reflective of risks from SHS exposure for the approximately 9% of children in the US today who have asthma.[54] All children in this study were exposed to SHS so we may only generalize our findings to exposed children. However, the degree of exposure varied widely in our sample, and there was no evidence of curvilinear or threshold effects that might suggest a “safe” level of SHS exposure. In addition, National data suggest that more than half of children are exposed to SHS.[55] Our sleep data were derived from parent report only. Additional study in this area should include use of child reported sleep problems and additional measures of sleep patterns such as polysomnography, actigraphy, or detailed sleep diaries. Finally, we had no information on prematurity in this sample which could be an important contributor to sleep problems.[56, 57]

Conclusion

Among children with asthma, exposure to second hand smoke negatively impacts sleep as evidenced by greater sleep onset delays, more frequent parasomnias, more sleep disordered breathing, increased daytime sleepiness, and greater overall sleep disturbance. The

consequences of inadequate sleep in children are not trivial. Sleep disturbances have been linked with increased behavior problems,[2-5] mental health problems,[1, 6] and poor school performance[1] in children. In addition, effects of poor sleep in childhood can persist into adulthood in the form of obesity[8, 9] and behavior and mood disorders.[7] Reduction in SHS exposure is an area with the potential for significant impact for physical and emotional health and as well as school performance among the pediatric population.

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Table 1

Sample Characteristics (N=219)

Characteristic	n (%) or mean ± sd (unless otherwise noted)
Age in years	9.4 ± 1.8
Male	134 (61.2%)
Race	
African American	122 (55.7%)
Caucasian	92 (42.0%)
Other	5 (2.3%)
Parent Education	
High school graduate or less	143 (65.3%)
Any college	76 (34.7%)
Parent Marital Status	
Married / living with someone	91 (41.6%)
Divorced / separated/ windowed	37 (16.8%)
Single – Never Married	91 (41.6%)
Household Income (median and inter-quartile range)	$25K ($15K, $45K)
Asthma Severity	
Mild	51 (23.2%)
Moderate	105 (48.0%)
Severe/Very Severe	63 (28.8%)
Maternal Smoking During Pregnancy	
None	73 (33.3%)
Until Pregnancy recognition	32 (14.6%)
Throughout Pregnancy	114 (52.1%)
Cigarettes Smoked in the Home Daily (median and inter-quartile range)	13 (9, 20)
Serum Cotinine (ng/mL, geometric mean and 95% confidence interval)	1.16 (0.10, 13.07)
Serum Cotinine (ng/mL, median and inter-quartile range)	1.45 (0.56, 2.69)
HOME Scores	46.9 ± 8.1
Maternal Depression (BDI)	12.4 ± 9.9
BASC (within clinical range ≥70)	
Externalizing	41 (18.8%)
Internalizing	58 (26.6%)
Behavior Symptoms	46 (21.1%)
Sleep Pattern	
Bed resistance	8.9 ± 2.7
Sleep onset delay	1.6 ± 0.8
Sleep duration	4.4 ± 1.7
Sleep anxiety	5.7 ± 1.8
Night wakings	4.3 ± 1.3
Parasomnias	9.6 ± 2.1
Sleep disordered breathing	4.2 ± 1.4

Characteristic	n (%) or mean ± sd (unless otherwise noted)
Daytime sleepiness	15.5 ± 3.3
Total sleep disturbance	51.6 ± 8.2
Total sleep disturbance (raw score ≥41)	203 (92.7%)

Table 2

Associations Between Serum Cotinine (independent variable) and Children's Sleep (dependent variable) Using Logistic and Multiple Regression (N=219)

Sleep Scale (cut point)	Bi-variate Association between Serum Cotinine and Sleep Outcomes			Full Model of Association between Serum Cotinine and Sleep Outcomes Including All Covariates			Final Model of Association between Serum Cotinine and Sleep Outcomes Including Significant Covariates		
	Odds Ratio or β*	95% CI	p	Odds Ratio or β*	95% CI	p	Odds Ratio or β*	95% CI	p
Bed Resistance (≥10)	1.29	1.09 – 1.53	**0.003**	1.34	0.95 – 1.88	0.093	1.21	0.995 - 1.47	0.056
Sleep Onset Delay (≥2)	1.07	0.92 – 1.24	0.39	1.50	1.08 - 2.06	**0.014**	1.53	1.15 - 2.03	**0.004**
Sleep Duration (≥5)	1.00	0.86 – 1.17	0.98	1.19	0.87 -1.63	0.27	1.12	0.92 – 1.37	0.24
Sleep Anxiety (≥7)	1.28	1.07 – 1.53	**0.006**	0.90	0.65 – 1.24	0.51	1.06	0.81 – 1.38	0.68
Night Wakings (≥5)	1.09	0.93 – 1.28	0.28	1.10	0.81 – 1.50	0.54	1.09	0.92 – 1.31	0.32
Parasomnias (≥12)	1.62	1.23 – 2.12	**0.0005**	1.91	1.09 – 3.34	**0.023**	1.95	1.36 – 2.79	**0.0002**
Sleep Disordered Breathing (≥5)	1.29	1.08 – 1.54	**0.005**	1.33	0.94 – 1.89	0.11	1.26	1.04 – 1.52	**0.02**
Daytime Sleepiness *	0.40	0.15 – 0.64	**0.002**	0.51	0.08 – 0.94	**0.021**	0.33	0.05 – 0.61	**0.022**
Total Sleep Disturbance *	1.40	0.81 – 1.99	**<0.0001**	1.42	0.37 – 2.47	**0.009**	1.14	0.54 – 1.74	**0.0002**

Age of child, gender, and asthma severity were included in all analyses. Other covariates were included if they were statistically relevant to the sleep scale of interest and included the following considerations: maternal race, marital status, education, income, maternal smoking during pregnancy, maternal depression, HOME inventory total score, household density (house volume/persons), number of siblings, use of asthma medication including short-acting inhaled medicines, long-acting inhaled medicines, and oral steroids.

* Multiple regression was used for daytime sleepiness and total sleep disturbance (β reported); all others used logistic regression on dichotomized sleep scales (OR reported).