Associations Between Secondhand Smoke Exposure and Sleep Patterns in Children
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Associations Between Secondhand Smoke Exposure and Sleep Patterns in Children

OBJECTIVES: The objective of this study was to investigate the relationship between exposure to secondhand smoke (SHS) and child sleep patterns among a group of children with asthma who were exposed regularly to tobacco smoke at home.

METHODS: We studied 219 children who were enrolled in an asthma intervention trial and were exposed regularly to SHS. Serum cotinine levels were used to measure exposure to tobacco smoke, and sleep patterns were assessed through parent reports using the Children’s Sleep Habits Questionnaire. Covariates in adjusted analyses included gender, age, race, maternal marital status, education, and income, prenatal tobacco exposure, maternal depression, Home Observation for Measurement of the Environment total score, household density, asthma severity, and use of asthma medications.

RESULTS: Exposure to SHS 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 SHS is associated with increased sleep problems among children with asthma. Pediatrics 2010;125:e261–e268
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, somatic complaints, behavior problems, and mental health problems. In addition, sleep problems during childhood are associated with increased incidence of anxiety and depression, aggressive behaviors, and attention problems in adulthood, which suggests a lasting impact of sleep problems on mental health. Poor childhood sleep also predicts the development of obesity and its associated morbidities, which indicates an important influence on health outcomes.

More than 25% of children experience some type of sleep problem during childhood. Among children with asthma, the prevalence of sleep problems is higher, with 40% to 60% having some difficulty. Children with asthma are nearly 4 times more likely to experience sleep-disordered breathing, which results in sleep disruption and decreased sleep efficiency, reduced sleep quality, increased nighttime activity levels, and more difficulties with daytime sleepiness. Sleep efficiency has been shown to improve with effective treatment of asthma symptoms, but even children with clinically stable asthma have worse sleep quality and more daytime sleepiness than do children without asthma.

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. Adult and adolescent smokers report more sleep problems, such as trouble initiating sleep, maintaining sleep, difficulty waking, and daytime sleepiness. 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, compared with pregnant women who do not smoke. Interestingly, women who are nonsmokers but are exposed to secondhand 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. This effect of SHS 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 and more symptoms of sleep-disordered breathing compared with those who are not exposed. Because tobacco smoke is a known contributor to asthma severity, 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. A major limitation of these studies linking SHS and sleep difficulties in children is the fact that they relied on parent reports of exposure, instead of more-precise and more-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 levels, to quantify exposure objectively and a validated pediatric sleep survey to characterize sleep patterns. We hypothesized that children with asthma who were exposed to higher levels of SHS would exhibit more sleep problems, as reported by parents, compared with children with lower levels of exposure.

METHODS

This study used the Cincinnati Asthma Prevention 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 before initiation of the asthma intervention.

Recruitment and enrollment procedures for the 6- to 12-year-old children are described in detail elsewhere. Briefly, all children in the sample had physician-diagnosed asthma that had been treated within the previous year and exposure to SHS from ≥5 cigarettes per day at home, according to parent report. Children were identified on the basis of hospital and clinic billing records, and parents of 1678 children were contacted for completion of a screening survey and request for participation, if eligible. Children were excluded if they had other respiratory diseases, heart disease, mental retardation, or 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 whether windows were open during car exposure. SHS exposure also was measured objectively by using serum cotinine levels detected in samples collected at the baseline home visit, which represent 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 to 72 hours. However, because of stability of exposure patterns over time, a one-time cotinine measurement is considered representative of typical daily exposure.34 Cotinine levels in serum were measured by the Centers for Disease Control and Prevention with published methods involving high-performance liquid chromatography linked to atmospheric-pressure chemical ionization-tandem mass spectrometry.35 We applied a logarithmic base 2 transformation for analysis of serum cotinine levels because of the skewed distribution of the raw data. This provides for 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 level for each doubling of the cotinine level.

The Children’s Sleep Habits Questionnaire (CSHQ)36 was used to measure child sleep patterns within the previous 2 weeks, as reported by the primary caregiver. The CSHQ yields a total sleep disturbance score and scores for 8 scales (ie, bedtime resistance, sleep-onset delay, sleep duration, sleep anxiety, night wakings, parasomnias, sleep-disordered breathing, and 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 ($\alpha = 0.78$ for a clinical sample), and test-retest reliabilities among the scales are high ($r = 0.62–0.79$). A total CSHQ score of $\geq 41$ has sensitivity of 0.80 and specificity of 0.72, properly classifying 80% of a group with clinically relevant sleep disorders. To ensure uniform thorough completion, this measure was administered in 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 because of small group sizes. Parent report of asthma symptoms is an effective means of characterizing child asthma and is not enhanced by asthma diaries or pulmonary function testing.36 Parents also reported asthma medication use by the child, including use of short-acting bronchodilators, long-acting inhaled steroids, and orally administered steroids prescribed for treatment of acute exacerbations.

Other measured covariates were maternal depression (Beck Depression Inventory II37 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 instrument 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 generated with SOLAS 3.0 (Statistical Solutions, Cork, Ireland), on the basis of age, race, and gender, were used. Univariate analyses involved inspection of frequencies and estimation of means and associated SDs. Because of nonnormal distributions, serum cotinine levels are reported as geometric means and 95% confidence intervals (CIs), and household income is reported as median and 25th and 75th percentile values. We used linear regression for the daytime sleepiness and total sleep disturbance scales. The distributions of the other sleep scales reflected varying degrees of nonnormality; therefore, responses were dichotomized, at approximately the 75th percentile value, and logistic regression was used. For each outcome measure, 3 models were developed, to reflect (1) the simple bivariate association between exposure and sleep, (2) the association after adjustment for all covariates (age, gender, race, maternal smoking during pregnancy, marital status, maternal education, household income, household density, 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, gender, and asthma severity were retained irrespective of statistical significance. Other covariates were retained if they accounted for significant variance on the given sleep scale ($P < .05$). Also, if removal of the covariate from the model was associated with a $>10\%$ change in the regression coefficient for serum cotinine levels, then it was retained in the model. SAS 9.1 (SAS Institute, Cary, NC) 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 black. Children in the sample were exposed to a median of 13 cigarettes per day in their homes, as
reported by their parents, and the geometric mean serum cotinine level for the sample was 1.16 ng/mL. The correlation between serum cotinine levels and parent-reported exposure was 0.39 (P < .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 et al.32 Internal consistency for the sleep measure also was comparable to the findings of Owens et al.32 Surprisingly, 93% of the children in the sample had a CSHQ total sleep disturbance score (≥41) that would be considered clinically relevant. The mean sleep time reported by parents was 9.6 hours per night.

In bivariate analyses, the associations between the logarithm of serum cotinine levels 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 levels.

In multivariate analyses including all potential covariates of child sleep patterns, we found that higher levels of SHS exposure were significantly associated with higher scores (ie, more problems) on the sleep-onset delay, parasomnias, daytime sleepiness, and total sleep disturbance scales. Final models, including only covariates that had a relationship with the sleep scale of interest or affected the coefficient for cotinine levels, 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 because of 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 nonmarried parent was associated with fewer parasomnias and increased daytime sleepiness. 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 nonmarried parent was associated with fewer parasomnias and increased daytime sleepiness. Decreased 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.

**TABLE 1 Sample Characteristics (N = 219)**

<table>
<thead>
<tr>
<th>Category</th>
<th>Mean ± SD</th>
<th>Median (interquartile range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean ± SD, y</td>
<td>9.4 ± 1.8</td>
<td>9.4 (8–11)</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>134 (61.2)</td>
<td></td>
</tr>
<tr>
<td>Race, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>122 (55.7)</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>92 (42.0)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>5 (2.3)</td>
<td></td>
</tr>
<tr>
<td>Parent education, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school graduate or less</td>
<td>143 (65.3)</td>
<td></td>
</tr>
<tr>
<td>Any college</td>
<td>76 (34.7)</td>
<td></td>
</tr>
<tr>
<td>Parent marital status, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married or living with someone</td>
<td>91 (41.6)</td>
<td></td>
</tr>
<tr>
<td>Divorced, separated, or widowed</td>
<td>37 (16.8)</td>
<td></td>
</tr>
<tr>
<td>Single, never married</td>
<td>91 (41.6)</td>
<td></td>
</tr>
<tr>
<td>Household income, median (interquartile range), $</td>
<td>25 000 (15 000–45 000)</td>
<td></td>
</tr>
<tr>
<td>Asthma severity, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>51 (23.2)</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>105 (48.0)</td>
<td></td>
</tr>
<tr>
<td>Severe/very severe</td>
<td>63 (28.8)</td>
<td></td>
</tr>
<tr>
<td>Maternal smoking during pregnancy, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>73 (33.3)</td>
<td></td>
</tr>
<tr>
<td>Until pregnancy recognition</td>
<td>32 (14.6)</td>
<td></td>
</tr>
<tr>
<td>Throughout pregnancy</td>
<td>114 (52.1)</td>
<td></td>
</tr>
<tr>
<td>No. of cigarettes smoked in home daily, median (interquartile range)</td>
<td>13 (9–20)</td>
<td></td>
</tr>
<tr>
<td>Serum cotinine level</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Geometric mean (95% CI), ng/mL</td>
<td>1.16 (0.10–13.07)</td>
<td>1.16 (0.10–13.07)</td>
</tr>
<tr>
<td>Median (interquartile range), ng/mL</td>
<td>1.45 (0.56–2.69)</td>
<td></td>
</tr>
<tr>
<td>HOME score, mean ± SD</td>
<td>46.9 ± 8.1</td>
<td></td>
</tr>
<tr>
<td>Maternal depression score, mean ± SD</td>
<td>12.4 ± 9.9</td>
<td></td>
</tr>
<tr>
<td>Behavior Assessment System for Children-2 score within clinical range</td>
<td>41 (18.8)</td>
<td></td>
</tr>
<tr>
<td>Externalizing symptoms</td>
<td>58 (26.8)</td>
<td></td>
</tr>
<tr>
<td>Internalizing symptoms</td>
<td>46 (21.1)</td>
<td></td>
</tr>
<tr>
<td>Behavior symptoms</td>
<td></td>
<td></td>
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<tr>
<td>Sleep pattern score, mean ± SD</td>
<td></td>
<td></td>
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<tr>
<td>Bed resistance</td>
<td>8.9 ± 2.7</td>
<td></td>
</tr>
<tr>
<td>Sleep-onset delay</td>
<td>1.6 ± 0.8</td>
<td></td>
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<tr>
<td>Sleep duration</td>
<td>4.4 ± 1.7</td>
<td></td>
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<tr>
<td>Sleep anxiety</td>
<td>5.7 ± 1.8</td>
<td></td>
</tr>
<tr>
<td>Night waking</td>
<td>4.3 ± 1.3</td>
<td></td>
</tr>
<tr>
<td>Parasomnias</td>
<td>9.6 ± 2.1</td>
<td></td>
</tr>
<tr>
<td>Sleep-disordered breathing</td>
<td>4.2 ± 1.4</td>
<td></td>
</tr>
<tr>
<td>Daytime sleepiness</td>
<td>15.5 ± 3.3</td>
<td></td>
</tr>
<tr>
<td>Total sleep disturbance</td>
<td>51.6 ± 8.2</td>
<td></td>
</tr>
<tr>
<td>Total sleep disturbance raw score of ≥41, n (%)</td>
<td>203 (92.7)</td>
<td></td>
</tr>
</tbody>
</table>
We found cotinine level-gender interactions for the sleep-onset delay (odds ratio [OR]: 0.63; \( P = .011 \)) and sleep anxiety (OR: 1.47; \( P = .05 \)) scales; therefore, we performed gender-stratified regression analyses for 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 levels and higher scores for sleep anxiety (OR: 1.54; \( P = .003 \)). For girls, a statistically significant relationship was found between serum cotinine levels and sleep-onset delay (OR: 1.54; \( P = .008 \)).

**DISCUSSION**

For 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 gender-cotinine level interactions. In regression analyses stratified according to gender, greater exposure to SHS was associated with greater sleep anxiety in boys and greater sleep-onset delay in girls.

SHS exposure was associated with increased incidence of parasomnias in this sample of children. Parasomnias reflect partial arousal from either non-rapid eye movement or rapid eye movement sleep and, although usually benign, can be highly distressing to children and their families. More than 80% of preschoolers experience parasomnias, but their incidence decreases with age.39 Adult men who smoke report more nightmares and disturbing dreams than do 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-old youths. In contrast to our findings, however, they have been reported more frequently among girls (72%) than boys (55%).40 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.41 No other studies have investigated the relationship between SHS and sleep-onset delays among children.

The exact mechanism through which SHS exposure may affect children's sleep is not clear. We briefly explore 3 possible explanations, that is, 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,24,42 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 disturbances among children with asthma.

Although adolescent and adult smokers report disturbances in sleep,22–24,41,43 a clear causal relationship between to-
bacco and sleep disorders has been difficult to establish. 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. Nicotine is associated with altered sleep architecture, resulting in reduced sleep time and efficiency, longer sleep latency, lighter sleep, and reduced rapid eye movement sleep. Frequent night wakings among smokers often are 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 rapid eye movement sleep. Colrain et al reported increased night wakings and sleepiness as the most-consistent findings of research on smoking cessation. Insomnia, sleep disturbance, and anxiety also are symptoms of nicotine withdrawal listed in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. 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, which results 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 presented evidence that newborns who were exposed prematurely to tobacco experienced nicotine withdrawal shortly after birth. 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, pregnant women, and preschool-aged children, which indicates that even small amounts of exposure to nicotine, as would occur in SHS exposure, are sufficient to affect sleep adversely. Our study contributes additional evidence that SHS affects sleep among school-aged children with asthma.

This study is not without limitations. All children in the study had asthma, and the results may not be generalizable to populations of children without asthma. However, these results may be reflective of risks from SHS exposure for the 9% of children in the United States today who have asthma. All children in this study were exposed to SHS, and we can generalize our findings only to exposed children. 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 one half of children are exposed to SHS. Our sleep data were derived from parent reports 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.

CONCLUSIONS

Among children with asthma, exposure to SHS affects sleep negatively, 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, mental health problems, and poor school performance in children. In addition, effects of poor sleep in childhood can persist into adulthood in the form of obesity and behavior and mood disorders. Reduction in SHS exposure is an area with the potential for significant impact for physical and emotional health and school performance in the pediatric population.

ACKNOWLEDGMENTS

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