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Changes in Environmental Tobacco Smoke Exposure and Asthma Morbidity Among Urban School Children

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Abstract

Background: Environmental tobacco smoke (ETS) exposure is associated with poor asthma outcomes in children. However, little is known about natural changes in ETS exposure over time in children with asthma and how these changes may affect health-care utilization. This article documents the relationship between changes in ETS exposure and childhood asthma morbidity among children enrolled in a clinical trial of supervised asthma therapy.

Methods: Data for this analysis come from a large randomized clinical trial of supervised asthma therapy in which 290 children with persistent asthma were randomized to receive either usual care or supervised asthma therapy. No smoking cessation counseling or ETS exposure education was provided to caregivers; however, children were given 20 min of asthma education, which incorporated discussion of the avoidance of asthma triggers, including ETS. Asthma morbidity and ETS exposure data were collected from caregivers via telephone interviews at baseline and at the 1-year follow-up.

Results: At baseline, 28% of caregivers reported ETS exposure in the home and 19% reported exposure outside of the primary household only. Among children whose ETS exposure decreased from baseline, fewer hospitalizations ($p = 0.034$) and emergency department (ED) visits ($p \leq 0.001$) were reported in the 12 months prior to the second interview compared to the 12 months prior to the first interview. Additionally, these children were 48% less likely ($p = 0.042$) to experience an episode of poor asthma control (EPAC).

Conclusions: This is the first study to demonstrate an association between ETS exposure reduction and fewer EPACs, respiratory-related ED visits, and hospitalizations. These findings emphasize the importance of ETS exposure reduction as a mechanism to improve asthma control and morbidity. Potential policy implications include supporting ETS reductions and smoking cessation interventions for parents and caregivers of children with asthma. Research to identify the most cost-effective strategy is warranted.

Trial registration: Clinicaltrials.gov Identifier: NCT00110383

Keywords

asthma; children; environmental tobacco smoke; tobacco smoke pollution

The Centers for Disease Control and Prevention reports¹ that 60% of children aged 3 to 11 years in the United States are exposed to environmental tobacco smoke (ETS) based on serum cotinine levels. No other age group of nonsmokers has a higher proportion of exposure, of subjects living with at least one smoker, or median cotinine concentrations than the 3- to 11-year-old age group.² Exposure is particularly high in low-income and non-Hispanic black children.^{1,3} Twenty-five percent of children live with at least one smoker,¹ and parental smoking accounts for 90% of children's exposure in the home.³ Over time, ETS exposure in nonsmokers has decreased significantly; however, this decline has been smaller in children than adults.²

In all children, ETS exposure is associated with increased risk of sudden infant death syndrome, respiratory infections, and otitis media.² ETS exposure is associated with asthma prevalence and airway irritation in children with asthma.⁴⁻¹¹ Furthermore, strong evidence exists that ETS exposure is associated with poor asthma outcomes including increased asthma symptoms,^{8, 12-14} illness-related school absences,^{15,16} and health-care use,¹⁶ as well as decreased lung function.^{12,14,16} In fact, ETS exposure may account for up to 1,000,000 childhood asthma exacerbations a year.¹⁷

ETS exposure reduction is a fairly new area of scientific study. A recent Cochrane review of the effectiveness of interventions aimed at reducing ETS exposure in children indicated that few produced a statistically significant reduction.¹⁷ However, it appears that interventions that employ intensive counseling may be more effective than brief interventions.¹⁸ Winickoff et al^{19,20} report significant changes in parental smoking behaviors in a study of counseling and nicotine replacement. However, long-term smoking cessation rates were not reported and ETS exposure was not measured.

Information linking changes in ETS exposure with changes in health-care utilization among children with asthma is limited. Wilson et al²¹ demonstrated that an intense ETS reduction intervention with individual feedback in families of children with asthma reduced asthma-related urgent health-care utilization. However, reductions in hospitalizations and emergency department (ED) visits were not statistically significant, possibly due to the limited sample size. Furthermore, the difference in urine cotinine levels between the intervention and control groups was not significant. Health-care utilization was obtained via claims data; however, urine cotinine required follow-up visits where significant attrition was observed (only 59% completion).

Little is known about natural changes in ETS exposure in children with asthma over time and how this may affect health-care utilization. This article documents the relationship between changes in ETS exposure and childhood asthma morbidity in a clinical trial of supervised asthma therapy in urban elementary schools.

Materials and Methods

Data for this analysis come from a large randomized trial^{22,23} of supervised asthma therapy that has been previously described in detail. Briefly, children were eligible if they met the following criteria: physician-diagnosed persistent asthma requiring daily controller medication, enrollment in 1 of 36 participating schools, and ability to use a dry-powder inhaler and a peak flowmeter. Children were recruited through local schools, physician offices, and health department clinics and randomized to either usual care or supervised therapy where

controller use (budesonide; Pulmicort Turbuhaler; AstraZeneca; Wilmington, DE) was directly observed at school. Controller and quick-relief medication were provided at no cost. No smoking cessation counseling or ETS exposure education was provided to caregivers; however, children were given 20 min of asthma education that incorporated discussion on the avoidance of asthma triggers including ETS.

Two hundred ninety children were randomized, and 240 children (83%) completed the main study. Transfer to a nonparticipating school accounted for 98% of the attrition, and attrition did not differ between treatment groups ($p = 0.26$). Asthma morbidity and ETS exposure were collected from caregivers via telephone interviews at baseline (August 2005) and at follow-up (August 2006). Two hundred sixty-five family caregivers completed the first interview; 256 caregivers (97%) completed both interviews. Caregivers were asked to self-report the child's respiratory-related ED and hospital visits in the prior 12 months. The distribution of ED visits and hospitalizations was highly skewed; therefore, data were collapsed into two categories (0 and ≥ 1). ETS exposure was assessed using two questions: (1) "Are there smokers in the house where your child lives?" and (2) "Are there smokers in other places where your child spends a lot of time, such as day care or a friend's house?" ETS exposure was grouped into three categories: (1) no exposure; (2) exposure outside of the primary household only; or (3) exposure inside the primary household. The number of children who were exposed both in and out of the primary household (21 of 265 children at baseline) was small and therefore were included in the third and highest ETS exposure category. The trend in ETS exposure between the first and second interviews was categorized as follows: remained the same; increased (*eg*, none at baseline but outside of home or inside home at the second interview); or decreased (*eg*, inside the home at baseline but outside of home or none at the second interview).

An episode of poor asthma control (EPAC) was defined as one or more of the following each month: (1) an absence from school due to respiratory illness/asthma; (2) average use of quick relief medication more than two times per week (not including preexercise treatment); or (3) at least one red or yellow peak flowmeter reading. Absences and peak flowmeter readings were collected daily through use of an Internet-based data collection system.²⁴ Quick relief medication use was measured through the use of a doser (Doser; Meditrack; Easton, MA) attached to the child's inhaler that activated automatically to record each use.

The University of Alabama at Birmingham Institutional Review Board and an independent three-member Data Safety and Monitoring Board approved and monitored the study. All parents provided informed consent and children provided assent.

Statistical Analysis

There were no differences in baseline characteristics between those children whose caregiver completed both interviews and those for whom only one was completed; therefore, this analysis reports on the results from caregivers who completed both interviews ($n = 256$). Furthermore, the trends in ETS exposure between the two interview periods did not differ between the usual care and supervised therapy groups ($p = 0.22$); therefore, the groups were combined in this analysis. Means and proportions were used to examine the demographic characteristics of the study population. A χ^2 test of association was used to determine whether the trend in ETS exposure between the first and second interviews differed by treatment group. McNemar test (2×2 tables) or Bowker test (3×3 tables) were utilized to determine whether the proportion of children in each level of ETS exposure was similar across interviews.²⁵ Further, χ^2 tests of association were used to assess whether differences in health-care utilization existed among ETS exposure groups. Logistic regression was used to examine the relationship between the change in ETS exposure and the likelihood of experiencing at least one EPAC. We examined interactions with year and intervention group, in order to assess whether the relationship between change in ETS exposure and the likelihood of experiencing an EPAC differed by

levels of these factors. In addition, we controlled for the proportion of budesonide refills at baseline and the change in the proportion of refills between the baseline and intervention periods. Results were determined to be statistically significant if the *p* value was < 0.05.

Results

Table 1 shows the demographic characteristics of the sample at baseline (*n* = 256). The mean age of children was 11 years (SD, 2.1 years). Fifty-eight percent of children (*n* = 149) were male, and 90% (*n* = 230) were black. Eighty percent of the children had moderate persistent asthma. At baseline, 28% of caregivers reported ETS exposure in the home and 19% reported exposure outside of the primary household only. There were no statistically significant differences at baseline among children in the three smoke exposure categories.

Table 2 shows ETS exposure at the baseline and follow-up interviews. Seventy-four percent (*n* = 190) of caregivers reported no change in the child's ETS exposure at the second interview, 17% (*n* = 44) reported less exposure, and 9% (*n* = 22) reported increased exposure. At the second interview, a greater proportion of children were reported to have no ETS exposure (*p* = 0.047).

Among children whose ETS exposure decreased, fewer hospitalizations (*p* = 0.034) and ED visits (*p* < 0.001) were reported in the 12 months prior to the second interview as compared to the 12 months prior to the first interview (Figs 1, 2). Additionally, the odds of these children experiencing an episode of poor asthma control are 45% of those for children with no change or increased ETS exposure (odds ratio [OR], 0.45; 95% confidence interval [CI], 0.23 to 0.88; *p* = 0.019). No significant interaction between change in ETS exposure and either year or intervention group was detected. Adjusting for the change in the proportion of refills between the baseline and intervention periods only slightly attenuated the observed relationship indicating that the observed effect of ETS exposure on episodes of poor asthma control cannot be accounted for by a change in use of inhaled steroids. When controlling for this variable, the odds of having an episode of poor asthma control among children whose exposure decreased were 48% of the odds of having an episode of poor asthma control among children with no change or increased ETS exposure (OR, 0.48; 95% CI, 0.26, *p* = 0.042).

Discussion

This is the first study to demonstrate a statistically significant association between ETS exposure reduction and fewer episodes of poor asthma control, respiratory-related ED visits, and hospitalizations. Secondhand smoke exposure within this population of urban school children with asthma was 47%. Exposure in the primary home accounted for 60% of the exposure. Over time, ETS exposure decreased from 47 to 40% among the children enrolled in this clinical trial. Children who had any decrease in ETS exposure had fewer episodes of poor asthma control, ED visits and hospitalizations than those who had the same or increased exposure. This demonstrates the potential importance of ETS exposure reduction as a mechanism to improve asthma control and morbidity.

ETS exposure reduction is a fairly new area of scientific study. A Cochrane review²⁶ of the ETS reduction interventions indicated that few programs produced a statistically significant reduction. Behavioral interventions that employ more intensive strategies are more effective than those that are less intensive,^{2,18} and a combination approach that includes both counseling and pharmacologic therapy is more effective than either alone.² To date, only two studies by Winickoff et al^{19,20} report on a combination approach among smoking parents of children. In these studies, Winickoff et al^{19,20} report that the number of quit attempts among parents increased and the number of cigarettes smoked in the house and car decreased following the

intervention. The 7-day abstinence rates at 2 months in these studies were 18%²⁰ and 21%,¹⁹ respectively. Behavioral changes noted include fewer smoked cigarettes inside the home, the car, and in the presence of the child.^{19,20} These studies indicate that ETS exposure reduction among children is feasible; however, this has yet to be linked to changes in asthma outcomes or health care utilization.

The observed reductions in health-care utilization in this study may have significant implications for the economic feasibility of ETS reduction strategies. While ED visits and hospitalizations are relatively uncommon, they are expensive. Therefore, costs associated with ETS reduction strategies could be offset by lower health expenditures in the child. Given that the majority of ETS exposure in the home is due to parental smoking, the most effective ETS reduction strategy may be to provide smoking cessation interventions to parents and possibly other household members. Additionally, smoking cessation has the potential to not only reduce ETS-related health expenditures in the child with asthma but to also offer direct health benefits to nonasthmatic siblings and the smoker himself. The combination of these effects may make smoking cessation interventions the most cost-effective ETS reduction strategy.

Another important finding of this study is that reduction in ETS exposure leads to fewer episodes of poor asthma control that do not necessarily lead to ED visits and hospitalizations. Improved asthma control may increase the number of asthma symptom-free days and improve quality of life. It may also lead to fewer medication needs and urgent health-care visits. If realized, these benefits would make ETS reduction and smoking cessation interventions in parents of children with asthma even more cost-effective. Future studies should examine the cost-effectiveness of providing intensive ETS reduction strategies in combination with pharmacotherapy.

The percentage of children who had at least one ED visit at baseline was higher among those who had a decrease in ETS exposure than the percentage observed in children who did not have an ETS reduction. One explanation is that parents obtained smoking cessation or ETS reduction counseling at the time of the ED visit and complied with the advice. This pattern was also observed in the hospitalization data. If this is true, it may reinforce the notion that ED visits and hospitalizations represent a window of opportunity for intervention as many parents are motivated to quit.^{27,28}

One limitation of this study is that the child's ETS exposure was collected via caregiver report. The disadvantage of this method is the potential for inaccurate reporting, particularly underreporting of the child's exposure. Measures of tobacco metabolites obtained from samples of bodily fluids are often used as objective measures of ETS exposure in children. While these measures are considered to be less subjective, there are a number of limitations to their use including cost and difficulty of obtaining specimens. Parental report and biological samples are correlated; however, Hovell et al²⁹ suggest that parental reports should be confirmed with direct measures of ETS exposure. Emerson et al³⁰ examined the accuracy of ETS exposure measures among asthmatic children and found the reliability estimates for parent-reported tobacco use to be much greater than that of urine cotinine assays. In fact, the reliability of urinary cotinine was 0.04, indicating that the measure was unreliable. Therefore, future work should examine this question prospectively using both parental report and measures of tobacco metabolites as markers of ETS exposure in the children.

Another limitation of this analysis is that the number of children who had a hospitalization or ED visit for asthma was small. These are relatively uncommon events even among children with asthma, and future studies should be powered to detect these differences.

Potential policy implications from these findings include the importance of identifying funding mechanisms to provide ETS reduction and smoking cessation counseling for parents and

caregivers of children with asthma. Research to identify the most cost-effective strategy is warranted. It is likely the most cost-effective strategy is a smoking cessation intervention that combines counseling with pharmacologic therapies. This combination approach has been deemed the most effective smoking cessation strategy² and offers potential health benefits to the child with asthma, nonasthmatic siblings, and the smoker himself.

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Abbreviations

ED, emergency department; EPAC, episode of poor asthma control; ETS, environmental tobacco smoke.

References

1. Children and secondhand smoke exposure: excerpts from the Health Consequences of Involuntary Exposure to Tobacco Smoke. a report of the Surgeon General. US Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; Atlanta, GA: 2007.
2. Public Health Service. Treating tobacco use and dependence. US Department of Health and Human Services, Public Health Service; Rockville, MD: 2008.
3. US Environmental Protection Agency. Fact sheet: national survey on environmental management of asthma and children's exposure to ETS. Available at: http://www.epa.gov/asthma/pdfs/survey_fact_sheet.pdf. Accessed October 19, 2008
4. Lanphear BP, Alinge CA, Auinger P, et al. Residential exposures associated with asthma in US children. *Pediatrics* 2001;107:505–511. [PubMed: 11230590]
5. Lewis SA, Antoniak M, Venn AJ, et al. Secondhand smoke, dietary fruit intake, road traffic exposures, and the prevalence of asthma: a cross-sectional study in young children. *Am J Epidemiol* 2005;161:406–411. [PubMed: 15718476]
6. Pattenden S, Antova T, Neuberger M, et al. Parental smoking and children's respiratory health: independent effects of prenatal and postnatal exposure. *Tob Control* 2006;15:294–301. [PubMed: 16885578]
7. Gergen PJ, Fowler JA, Maurer KR, et al. The burden of environmental tobacco smoke exposure on the respiratory health of children 2 months through 5 years of age in the United States: Third National Health and Nutrition Examination Survey, 1988–1994. *Pediatrics* 1998;101:1–6. [PubMed: 9417143]
8. Weitzman M, Gortmaker SL, Walker DK, et al. Maternal smoking and childhood asthma. *Pediatrics* 1990;85:505–511. [PubMed: 2314963]
9. Cook DG, Strachan D. Summary of effects of parental smoking on the respiratory health of children and implications for research. *Thorax* 1999;54:357–366. [PubMed: 10092699]
10. Strachan DP, Cook DG. Health effects of passive smoking: parental smoking and childhood asthma; longitudinal and case control studies. *Thorax* 1998;53:204–212. [PubMed: 9659358]
11. Martinez FD, Cline M, Burrows B. Increase incidence of asthma in children of smoking mothers. *Pediatrics* 1992;89:21–26. [PubMed: 1728015]
12. Schwartz J, Timonen KL, Pekkanen J. Respiratory effects of environmental tobacco smoke in a panel study of asthmatic and symptomatic children. *Am J Respir Crit Care Med* 2000;161:802–806. [PubMed: 10712325]
13. Sturm JJ, Yeatts K, Loomis D. Effects of tobacco smoke exposure on asthma prevalence and medical care use in North Carolina middle school children. *Am J Public Health* 2004;94:308–313. [PubMed: 14759947]

14. Chilmonczyk BA, Salmunm LM, Megathlin KN, et al. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. *N Engl J Med* 1993;328:1665–1669. [PubMed: 8487825]
15. Gilliland FD, Berhane K, Wenten IS, et al. Environmental tobacco smoke and absenteeism related to respiratory illness in schoolchildren. *Am J Epidemiol* 2003;157:861–869. [PubMed: 12746237]
16. Mannino DM, Homa D, Redd S. Involuntary smoking and asthma severity in children: data from the Third National Health And Nutrition Examination Survey. *Chest* 2007;122:409–415. [PubMed: 12171810]
17. US Environmental Protection Agency. Respiratory health effects of passive smoking: lung cancer and other disorders. Environmental Protection Agency, Office of Research and Development, Office of Air and Radiation; Washington, DC: 1992.
18. Gehrman CA, Hovell MF. Protecting children from environmental tobacco smoke (ETS) exposure: a critical review. *Nicotine Tob Res* 2003;5:289–301. [PubMed: 12791524]
19. Winickoff JP, Hillis VJ, Palfrey JS, et al. A smoking cessation intervention for parents of children who are hospitalized for respiratory illness: the Stop Tobacco Outreach Program. *Pediatrics* 2003;111:140–145.
20. Winickoff JP, Buckley VJ, Palfrey JS, et al. Intervention with parental smokers in an outpatient pediatric clinic using counseling and nicotine replacement. *Pediatrics* 2003;112:1127–1133. [PubMed: 14595057]
21. Wilson SR, Yamada EG, Sudhaker R, et al. A controlled trial of an environmental tobacco smoke reduction intervention in low-income children with asthma. *Chest* 2001;120:1709–1722. [PubMed: 11713157]
22. Gerald LB, McClure LA, Harrington K, et al. Design of the supervised asthma therapy study: implementing an adherence intervention in urban elementary schools. *Contemp Clin Trials* 2008;29:304–310. [PubMed: 17804302]
23. Gerald LB, McClure LA, Mangan J, et al. Increasing adherence to inhaled steroids among school children: a randomized controlled trial of school based supervised asthma therapy. *Pediatrics*. 2009;in press
24. Mangan JM, Gerald LB. Asthma agents: monitoring asthma in school. *J School Health* 2006;76:300–302. [PubMed: 16918859]
25. Argresti, A. Categorical data analysis. John Wiley and Sons; New York, NY: 1990.
26. Roseby R, Waters E, Polnay A, et al. Family and carer smoking control programs for reducing children's exposure to environmental tobacco smoke. *Cochrane Database Syst Rev*. 2002;(3)database online
27. Mahabee-Gittens M. Smoking in parents of children with asthma and broncholitis in a pediatric emergency department. *Pediatr Emerg Care* 2002;18:4–7. [PubMed: 11862128]
28. DePue JD, McCabe B, Kazura A, et al. Assessment of parents' smoking behaviors at a pediatric emergency department. *Nicotine Tob Res* 2007;9:33–41. [PubMed: 17365734]
29. Hovell MF, Zakarian JM, Wahlgren DR, et al. Reported measures of environmental tobacco smoke exposure: trials and tribulations. *Tob Control* 2000;9(suppl):iii22–iii28. [PubMed: 10982901]
30. Emerson JA, Hovell MF, Meltzer SB, et al. The accuracy of environmental tobacco smoke exposure measures among asthmatic children. *J Clin Epidemiol* 1995;48:1251–1259. [PubMed: 7561987]

Figure 1.
Percentage of children with at least one hospitalization as categorized by change in ETS exposure.

Figure 2.
Percentage of children with at least one ED visit as categorized by change in ETS exposure.

Demographic Characteristics of Participants According to ETS Exposure at Baseline Among Those Completing Both Interviews*

Table 1

Characteristics	No Exposure (n = 135)	Outside Primary Household Only (n = 49)	Inside Primary Household (n = 72)	Total (n = 256)	p Value [†]
Age, yr	11.0 ± 2.2	10.6 ± 2.0	11.0 ± 1.9	11.0 ± 2.1	0.39
Male gender	81 (60)	31 (63)	37 (51)	149 (58)	0.36
Black race	121 (90)	47 (96)	62 (86)	230 (90)	0.21
Asthma severity (two missing)					
Mild persistent	17 (13)	12 (25)	9 (13)	38 (15)	0.26
Moderate persistent	111 (83)	35 (71)	56 (79)	202 (80)	
Severe persistent	6 (4)	2 (4)	6 (8)	14 (5)	
Rescue medication at school prior to study	17 (13)	9 (18)	12 (17)	38 (15)	0.55
Number of daily puffs of budesonide prescribed	2.7 ± 1.1	3.0 ± 1.1	2.8 ± 1.0	2.8 ± 1.0	0.16
Percentage of expected budesonide refills that were filled [‡]	0.59 ± 0.31	0.63 ± 0.29	0.52 ± 0.29	0.58 ± 0.30	0.14
≥ 1 hospitalization for asthma in past year	10 (7)	7 (14)	9 (13)	26 (10)	0.29
≥ 1 ED visit for asthma in past year	58 (43)	23 (47)	23 (32)	104 (41)	0.19
Average days absent	4.1 ± 3.9	3.2 ± 3.3	4.1 ± 3.4	3.9 ± 3.6	0.27
Average days absent due to respiratory illness	1.4 ± 2.4	0.83 ± 1.4	1.1 ± 2.2	1.2 ± 2.2	0.38

* Data are presented as mean ± SD or No. (%).

[†] All p values based on analysis of variance or χ^2 as appropriate.

[‡] Refill data calculated from enrollment through the end of the baseline period, defined as proportion of expected refills that were actually refilled.

Table 2

ETS Exposure at Baseline and Follow-up Interview (n = 256)*

Baseline Interview	Follow-up Interview			Total
	None	Outside Household Only	Inside Household	
None	117	11	7	135
Outside household only	26	19	4	49
Inside household	10	8	54	72
Total	153	38	65	256

* Data are presented as No.