# Is Secondhand Smoke Exposure Associated With Increased Exacerbation Severity Among Children Hospitalized for Asthma?

**AUTHORS**
Annie L. Andrews, MD, MSCR,[a] Nils Shirley, BS,[b] Elizabeth Ojukwu, BS,[c] Michelle Robinson, MS,[d] Michelle Torok, PhD,[d] Karen M. Wilson, MD, MPH[d]

[a]*Department of Pediatrics, Medical University of South Carolina, Charleston, South Carolina;*
[b]*Medical University of South Carolina College of Medicine, Charleston, South Carolina;*
[c]*University of Massachusetts Medical School Worcester, Massachusetts; and*
[d]*Department of Pediatrics, University of Colorado School of Medicine, Aurora, Colorado*

**KEY WORDS**
asthma exacerbation, admission

**ABBREVIATIONS**
CHCO: Children's Hospital Colorado
CI: confidence interval
CPAP: continuous positive airway pressure
ED: emergency department
IRR: incidence rate ratio
IV: intravenous
LOS: length of stay
MUSC: Medical University of South Carolina
OR: odds ratio
PNA: pulmonary nodular amyloidosis
RedCap: Research Electronic Data Capture
SHS: secondhand smoke

www.hospitalpediatrics.org
doi:10.1542/hpeds.2014-0128

Address correspondence to Annie L. Andrews, MD, MSCR, Medical University of South Carolina, Department of Pediatrics, 135 Rutledge Ave, MSC 561, Charleston, SC 29425. E-mail: andrewsan@musc.edu

HOSPITAL PEDIATRICS (ISSN Numbers: Print, 2154 - 1663; Online, 2154 - 1671).



**FINANCIAL DISCLOSURE:** The authors have indicated they have no financial relationships relevant to this article to disclose.

**FUNDING:** No external funding.

**POTENTIAL CONFLICT OF INTEREST:** The authors have indicated they have no potential conflicts of interest to disclose.

## abstract

**OBJECTIVE:** To determine the association between secondhand smoke (SHS) exposure and length of stay (LOS) and other exacerbation severity indicators in children hospitalized for asthma.

**METHODS:** We conducted a retrospective chart review at 2 children's hospitals. Patients aged 2 to 18 hospitalized for asthma in 2012 were included. Outcome variables included LOS, PICU, magnesium, and intravenous (IV) steroids. Bivariate analysis determined differences between SHS-exposed and non–SHS-exposed groups. Geometric means were used for LOS to account for skewed distribution. Logistic and zero-truncated negative binomial regression models were used to determine the independent association between SHS exposure and hospitalization severity indicators.

**RESULTS:** A total of 623 patients were included; 41% reported SHS exposure. Mean LOS was 47.5 hours. In the SHS-exposed group, LOS was 50.0 (95% confidence interval [CI] 46.7–54.0) and in the nonexposed group it was 45.8 (95% CI 43.4–48.4) ($P = .02$). In regression analysis, institution modified the effect of SHS exposure on LOS. At Children's Hospital Colorado, SHS exposure was associated with a 20% increase in LOS (incidence rate ratio 1.2, 95% CI 1.1–1.3). At the Medical University of South Carolina, there was no significant association. SHS-exposed patients were more likely to receive IV steroids (odds ratio 1.6, 95% CI 1.1–2.3)

**CONCLUSIONS:** Among children hospitalized for asthma, we identified a significant association at 1 institution between SHS exposure and LOS and found that IV steroid use was significantly associated with LOS at both institutions. Eliminating SHS exposure among children with asthma is important.

Asthma is the most prevalent chronic condition affecting children.[1] It is also one of the most frequent reasons for hospitalization among children.[1] Most children with asthma are exposed to secondhand smoke (SHS), and children from low-income families are more likely to be exposed.[2] Although SHS exposure has decreased for children in general, it remains stable in children with asthma.[3] SHS exposure has previously been associated with asthma severity based on symptom reporting, school absence, illness frequency, and lower forced expiratory volume in 1 second.[4] Additionally, SHS exposure is a risk factor for acute asthma exacerbations and associated emergency department (ED) visits, hospitalizations, and intubations.[5–8] Previous research has shown that a decrease in SHS exposure is associated with fewer ED visits and fewer hospitalizations among children with asthma.[9] Less is known about the effect of SHS exposure on severity of asthma

exacerbation once it is triggered and a child requires hospitalization.

SHS exposure is a modifiable risk factor. Therefore, if we are able to show that SHS exposure is associated with asthma exacerbation severity among hospitalized children, it will provide further evidence supporting the importance of smoking cessation interventions for family members of children with asthma. Successful caregiver smoking cessation could contribute to decreased resource use among children hospitalized for asthma and contribute to health care cost-savings.

The primary objective of this study was to determine if there is an association between SHS exposure and length of stay (LOS) in children hospitalized for asthma. The secondary objective was to determine if there is an association between SHS exposure and other markers of exacerbation severity among children hospitalized for asthma, including PICU admission, intravenous (IV) steroid use, and IV magnesium use.

## METHODS

We conducted a retrospective chart review at The Medical University of South Carolina Children's Hospital in Charleston, SC (MUSC), and Children's Hospital Colorado in Aurora, CO (CHCO). MUSC Children's Hospital is a tertiary care children's hospital with 186 beds and CHCO is a free-standing tertiary care children's hospital with 440 beds. Any patient 2 to 18 years of age with a primary discharge diagnosis of asthma (*International Classification of Diseases, Ninth Revision* codes 493.00–493.02, 493.10–493.12, 493.20–493.22, 493.8, and 493.9) who was hospitalized either at MUSC or CHCO between January 1, 2012, and December 31, 2012, was considered for inclusion. From this group with asthma, we eliminated patients with cystic fibrosis, active bronchopulmonary dysplasia, or tracheostomy. At the time of this study, MUSC used paper charts that are scanned and available electronically through an online application. CHCO's charts are electronic. A study team member at each site reviewed identified charts, and recorded data on data collection sheets by using random study identification numbers. This information was then recorded in Research Electronic Data Capture (RedCap). Study team members at both institutions had access to the RedCap file and could import data simultaneously. The RedCap file was imported directly into our statistical analysis software for analysis.

Information collected and recorded included demographics (age, gender, race/ethnicity, and insurance type). Clinical variables included comorbid conditions, such as prematurity and bronchopulmonary dysplasia, concurrent illnesses (specifically pneumonia or bronchiolitis), and home medications. Variables associated with current admission included admit day/time, discharge day/time, all diagnoses associated with admission, supplemental oxygen use, continuous positive airway pressure (CPAP) or EzPAP (a specific positive airway pressure device, similar to CPAP, used at CHCO; Smiths Medical, St Paul MN), intubation, PICU admission, IV steroid use, IV magnesium use, and other medications given either in the ED or inpatient setting. SHS exposure was assessed through chart review as well. SHS status was located in a variety of places, including the ED history and physical, the nursing intake assessment, or the admission history and physical. Previous research has shown that 93% of patients are screened for SHS exposure by at least 1 provider.[10] We considered any child with indoor or outdoor smoke exposure to be positive for SHS exposure. We also recorded a patient's smoking status when available, and these patients were included in the final analysis.

The primary outcome variable was LOS in hours. Additional secondary outcome variables were PICU admission, IV steroids, and IV magnesium. The primary independent variable was SHS exposure, which was treated as a dichotomous variable. The $\chi^2$, $t$ tests, and Wilcoxon rank sum tests were used to determine bivariate differences in demographic, clinical, and outcome variables between the SHS-exposed and non–SHS-exposed groups, as well as bivariate differences between institutions. Geometric means were used for LOS to account for skewed distribution.

Logistic and zero-truncated negative binomial regression models were used to determine the independent association between SHS exposure and hospitalization severity indicators, including LOS. Standard demographic variables were included as covariates in addition to variables that had significant associations with LOS in bivariate analysis. SAS 9.3 (SAS Institute, Inc, Cary, NC) was used for data analysis; $P < .05$ was considered to be statistically significant.

This study was approved by the institutional review board at both participating institutions.

## RESULTS

A total of 623 patients were included in the final analyses; 61% were male and 39% were black, 26% white, and 28% Hispanic. Median age was 6. Most

had public insurance. Overall, 41% of patients reported SHS exposure (with 9 patients reporting they themselves were smokers) (Table 1).

When comparing patients at each institution, there were statistically significant bivariate associations between institution and LOS, race/ethnicity, concurrent pneumonia diagnosis, use of CPAP/EzPAP, oxygen supplementation, and SHS exposure (Table 1).

The geometric mean LOS in the overall cohort was 47.5 hours (95% confidence interval [CI] 45.5–50.0). For those exposed to SHS, the geometric mean LOS was 50.0 hours (95% CI 46.7–54.0) and for the nonexposed patients, LOS was 45.8 hours (95% CI 43.4–48.4) ($P = .02$) (Table 2).

In addition to a difference in LOS, bivariate analysis revealed that SHS exposure status was also associated with IV steroid receipt, insurance status, and race/ethnicity. There were no bivariate associations between SHS exposure and PICU admission or IV magnesium use (Table 2).

During regression analysis for the outcome of LOS, it was noted that institution modified the association between SHS exposure and LOS. We ran a model with an interaction term between SHS and institution and the results were similar to the institution-specific models that are presented here. We chose to present the institution-specific models for this outcome variable for ease of interpretation. In zero-truncated negative binomial regression analysis after controlling for age, race/ethnicity, gender, insurance status, oxygen supplementation, home inhaled corticosteroid use, and concurrent pneumonia or bronchiolitis, SHS exposure was associated with a 20% increase in LOS (incidence rate ratio [IRR] 1.2, 95% CI 1.1–1.3) at CHCO but there was no statistically significant association between SHS and LOS at MUSC (IRR 1.0, 95% CI 0.8–1.4).

**TABLE 1** Demographic and Clinical Characteristics of Patients Admitted for Acute Asthma Exacerbation and Bivariate Comparisons by Institution

| Variable | Overall, $n = 623$ | CHCO, $n = 421$, 68% | MUSC, $n = 202$, 32% | *P* |
|---|---|---|---|---|
| SHS exposure, % | | | | .02 |
| Yes | 41 | 38 | 48 | |
| No | 59 | 62 | 52 | |
| Gender, % | | | | .33 |
| Male | 61 | 62 | 58 | |
| Female | 39 | 38 | 42 | |
| Race/ethnicity, % | | | | <.0001 |
| Black | 39 | 26 | 65 | |
| White | 26 | 26 | 24 | |
| Hispanic | 28 | 38 | 7 | |
| Other | 8 | 10 | 3 | |
| Age, median, y | 6.0 | 6.0 | 5.0 | .13 |
| Insurance, % | | | | .64 |
| Medicaid | 72 | 71 | 73 | |
| Private | 27 | 27 | 26 | |
| Self/other | 2 | 2 | 1 | |
| Prematurity, % | | | | .84 |
| Yes | 11 | 11 | 10 | |
| No/not documented | 89 | 89 | 90 | |
| History of asthma, % | | | | .57 |
| Yes | 76 | 75 | 77 | |
| No | 24 | 25 | 23 | |
| Taking ICS at home, % | | | | .80 |
| No | 50 | 51 | 50 | |
| Yes | 50 | 49 | 50 | |
| Concurrent PNA, % | | | | <.01 |
| No | 87 | 90 | 82 | |
| Yes | 13 | 10 | 18 | |
| Concurrent bronchiolitis, % | | | | .12 |
| No | 95 | 96 | 93 | |
| Yes | 5 | 4 | 7 | |
| PICU, % | | | | .36 |
| No | 81 | 80 | 83 | |
| Yes | 19 | 20 | 17 | |
| EzPAP or CPAP, % | | | | <.0001 |
| No | 88 | 82 | 100 | |
| Yes | 12 | 18 | 0 | |
| Oxygen, % | | | | <.0001 |
| No | 27 | 3 | 76 | |
| Yes | 73 | 97 | 24 | |
| Magnesium, % | | | | .95 |
| No | 77 | 77 | 77 | |
| Yes | 23 | 23 | 23 | |
| IV Steroids, % | | | | .38 |
| No | 71 | 72 | 68 | |
| Yes | 29 | 28 | 32 | |
| LOS geometric mean (95% CI) | 47.5 (45.5–50) | 48.6 (46.2–51.2) | 45.0 (43.4–48.4) | .02 |

ICS, inhaled corticosteroids.

**TABLE 2** Bivariate Analysis of SHS-Exposed Patients Compared With Non–SHS-Exposed Patients Among Children Admitted for Acute Asthma Exacerbation

| Variable | SHS Exposed, $n = 258$, 41% | Not SHS Exposed, $n = 365$, 59% | *P* |
|---|---|---|---|
| Gender, % | | | .77 |
| Male | 60 | 62 | |
| Female | 40 | 38 | |
| Race/Ethnicity, % | | | <.0001 |
| Black | 50 | 31 | |
| White | 26 | 25 | |
| Hispanic | 18 | 34 | |
| Other | 5 | 10 | |
| Age, median, y | 6.0 | 5.0 | .47 |
| Insurance, % | | | <.01 |
| Medicaid | 79 | 67 | |
| Private | 19 | 32 | |
| Self/other | 2 | 1 | |
| Prematurity, % | | | .17 |
| Yes | 13 | 9 | |
| No/not documented | 87 | 91 | |
| History of asthma, % | | | .18 |
| Yes | 79 | 74 | |
| No | 21 | 26 | |
| Taking ICS at home, % | | | .91 |
| No | 50 | 50 | |
| Yes | 50 | 50 | |
| Concurrent PNA, % | | | .71 |
| No | 88 | 87 | |
| Yes | 12 | 13 | |
| Concurrent bronchiolitis, % | | | .42 |
| No | 94 | 96 | |
| Yes | 6 | 4 | |
| PICU, % | | | .10 |
| No | 78 | 83 | |
| Yes | 22 | 17 | |
| EzPAP or CPAP, % | | | .55 |
| No | 87 | 89 | |
| Yes | 13 | 11 | |
| Oxygen, % | | | .19 |
| No | 29 | 25 | |
| Yes | 71 | 75 | |
| Magnesium, % | | | .30 |
| No | 75 | 78 | |
| Yes | 25 | 22 | |
| IV steroids, % | | | .01 |
| No | 65 | 75 | |
| Yes | 35 | 25 | |
| LOS, geometric mean (95% CI) | 50.0 (46.7–54.0) | 45.8 (43.4–48.4) | .02 |

ICS, inhaled corticosteroids.

In logistic regression analysis controlling for the same covariates with the addition of institution, SHS-exposed patients were more likely to receive IV steroids (odds ratio [OR] 1.6, 95% CI 1.1–2.3). There was a trend toward significance in the relationship between SHS exposure and PICU admission (OR 1.5, 95% CI 1.0–2.3). There was no significant association between SHS exposure and IV magnesium use (OR 1.3, 95% CI 0.9–2.0) (Table 3). For complete regression results see supplemental Table 4.

## DISCUSSION

Our study suggests that SHS exposure may contribute to asthma exacerbation severity among children admitted to the hospital. At CHCO, we found a statistically significant 20% increase in LOS among patients who report SHS exposure. In the overall cohort, we found a 60% greater likelihood of receiving IV steroids, and a trend toward an increased rate of PICU admission, both of which suggest more severe exacerbation. These data represent a previously undescribed health risk to children with asthma who are subjected to SHS exposure.

Previous research has shown that children with asthma who are exposed to SHS are more likely to experience acute exacerbations and require ED treatment, admission, and even intubation.[5,6,8] Our study contributes to the literature by showing that even once an exacerbation is triggered in a child with asthma and he or she requires hospitalization, those with SHS exposure may require longer hospitalization in some circumstances. Additionally, we found that SHS-exposed patients have a higher likelihood of receiving IV steroids, a more aggressive treatment typically reserved for the more acutely ill patients. Although there is no evidence that IV steroids are more effective than oral steroids, they can be used in patients with higher respiratory rates who cannot tolerate oral medications, and the National Heart, Lung, and Blood Institute Guidelines for the management of asthma recommend consideration of IV steroids for patients with an incomplete or poor response

**TABLE 3** Multivariable Analysis Results: Adjusted Relationship between SHS Exposure and Outcome Variables

| Outcome Variable | Overall Cohort | CHCO | MUSC |
|---|---|---|---|
| LOS, h,[a] IRR (95% CI) | 1.1 (1.0–1.2) | 1.2 (1.1–1.3) | 1.0 (0.8–1.1) |
| IV steroids,[b] OR (95% CI) | 1.6 (1.1–2.3) | 1.5 (0.9–2.5) | 1.8 (0.9–3.6) |
| PICU,[b] OR (95% CI) | 1.5 (1.0–2.1) | 1.4 (0.8–2.4) | 1.6 (0.5–2.7) |
| Magnesium,[b] OR (95% CI) | 1.3 (0.9–2.0) | 1.4 (0.9–2.3) | 1.0 (0.5–2.2) |

[a] The LOS model is a zero-truncated negative binomial regression model and included age, race, insurance, gender, oxygen use, home inhaled corticosteroids, diagnosis of pneumonia, and diagnosis of respiratory syncytial virus as covariates. The overall cohort model had significant interaction between institution and SHS in predicting LOS. An interaction term (institution*SHS) was included for this reason.
[b] Logistic regression models controlled for age, race, insurance, gender, oxygen use, home inhaled corticosteroids, diagnosis of pneumonia, diagnosis of respiratory syncytial virus, and institution (for the overall cohort model).

to therapy delivered in the ED.[11] The trend toward significance in the relationship between SHS exposure and PICU admission found in our data is consistent with findings published by van den Bosch et al in 2012.[12] In this multicenter retrospective case control study of 66 cases, the authors found that active or passive smoke exposure was independently associated with increased risk for PICU admission among children with asthma.[12] However, Lyell et al[13] found in a retrospective case control study of 52 cases in Australia that parental-reported smoking was not associated with PICU admission. It appears that the relationship between SHS exposure and risk for PICU admission has yet to be fully elucidated through these retrospective analyses. A prospective study that includes cotinine measurement would be an ideal way to answer this question.

Unlike other risk factors for severe asthma exacerbation, such as race/ethnicity, socioeconomic status, and history of prematurity, SHS exposure is a modifiable risk factor. Other modifiable risk factors include lack of usual source of care, noncompliance, and other environmental exposures. Children with asthma who require hospitalization are known to be high risk. They are more likely to require subsequent oral steroid bursts, ED visits, and hospitalizations.[14,15] If we can intervene while a child is in the hospital and decrease future SHS exposure, we may be able to significantly affect the disease course of the child with asthma. Winickoff et al[16] and Ralston et al[17] previously demonstrated the feasibility of smoking cessation interventions at the time a child is hospitalized for a respiratory illness. Our study provides further evidence that providers should consider capitalizing on this "teachable moment" when a parent is in the hospital with his or her child by delivering smoking cessation interventions.

The 20% increased LOS seen in patients exposed to SHS in the CHCO cohort equates to an ~10-hour longer hospital stay for each child with SHS exposure admitted for acute exacerbation. Although some may question the clinical significance of this finding, in the era of cost containment when many hospitals are focusing on discharge efficiency and early morning discharges, every additional hour in the hospital counts, and costs. The difference seen between patients at CHCO in Aurora, CO, and patients at MUSC in Charleston, SC, is likely, in part, because of differences in altitude and partial pressure of oxygen; 97% of the children admitted to CHCO required supplemental oxygen, whereas only 24% of children admitted to MUSC had the same requirement. It is plausible that the time it takes to wean children back to room air in Colorado contributes to the overall longer LOS and to the significant effect SHS exposure seems to have on LOS in that cohort that was not found in the MUSC cohort. There are institutional practice pattern differences that also may contribute to the LOS difference seen between the 2 institutions. Albuterol delivery is primarily via metered dose inhaler at CHCO and is given more often by nebulizer at MUSC. Additionally, albuterol weaning is driven by respiratory therapists at CHCO and by resident and attending physicians at MUSC. MUSC also has a step-down unit, whereas CHCO does not, which may affect PICU admission criteria and therefore may have affected our findings regarding the relationship between SHS exposure and PICU admission. However, both institutions use an albuterol-weaning pathway and prescribe prednisone/prednisolone (rather than dexamethasone) as their primary systemic steroid, and have the ability to give IV magnesium outside of the PICU. Other unmeasured variations in care also may contribute to the difference in LOS observed between these 2 institutions.

Our finding of increased likelihood of IV steroid receipt and a trend toward a significant increased risk of PICU admission among children with SHS exposure provides further evidence that among children who are hospitalized for acute asthma exacerbation, those who are exposed to SHS are at higher risk for more severe exacerbation.

There are several limitations to this study. We relied on caretaker reporting

of SHS exposure, which is not as reliable as cotinine measurements. A recent study by Howrylak et al[18] found high levels of underreporting of SHS exposure among hospitalized children when compared with serum and saliva levels. We were not able to assess for various exposure levels that may uniquely affect our outcome variables. This may have contributed to an underestimation of the actual effect of SHS exposure on exacerbation severity. However, a study that requires hair or blood sampling would be expensive and time-consuming. We feel that a retrospective chart review is an appropriate way to address our specific research question. In the future, researchers may wish to examine this relationship in further detail, and cotinine measurements would be an appropriate next step to advance knowledge in this area. Home inhaled corticosteroid use was included as a surrogate for disease severity. A more accurate measure of disease severity would have strengthened our analysis. Data on whether a patient had a usual source of care, which could contribute to disease control and exacerbation severity, was not available. Hospital system factors (eg, team availability to write discharge orders, families waiting for rides, or other barriers to patients physically leaving the hospital after discharge criteria are met) may affect discharge time and thus affect our primary outcome of LOS. These factors also may vary by institution, thus affecting our results. Of note, information on exact admit and discharge time is recorded electronically at both institutions, making this variable accurate and reliable. Our regression analysis should control for some of the variation in discharge time due to transportation issues by the inclusion of insurance status as a covariate. Future prospective studies may consider using the time a patient is medically ready for discharge as the primary outcome rather than actual discharge time. Additionally, as previously mentioned, asthma treatment practice patterns will vary by institution and therefore could contribute to the differences in LOS seen by institution. Because the medical records at the 2 institutions were different (paper versus electronic medical record), certain variables might be more accurately recorded or more easily discoverable at 1 institution when compared with the other. For example, oxygen supplementation was documented in several different areas of the MUSC charts, calling into question the accuracy of this variable at MUSC. On the other hand, information regarding oxygen supplementation was readily available in CHCO's electronic medical record.

In this study of children at 2 children's hospitals, there is a significant association between SHS exposure and LOS among children admitted for asthma at 1 institution. IV steroid use was more common among children exposed to SHS at both institutions, suggesting more severe exacerbation. Efforts to eliminate SHS exposure among children with asthma are needed, and smoking cessation interventions during the hospitalization may be a way to target parents of children at high risk with asthma.

## REFERENCES


1. Akinbami LJ, Moorman JE, Bailey C, et al. Trends in asthma prevalence, health care use, and mortality in the United States, 2001–2010. *NCHS Data Brief*. May 2012;(94):1–8.
2. Kit BK, Simon AE, Brody DJ, Akinbami LJ. US prevalence and trends in tobacco smoke exposure among children and adolescents with asthma. *Pediatrics*. 2013;131(3):407–414.
3. Quinto KB, Kit BK, Lukacs SL, Akinbami LJ. Environmental tobacco smoke exposure in children aged 3–19 years with and without asthma in the United States, 1999–2010. *NCHS Data Brief*. 2013;(126):1–8.
4. Mannino DM, Homa DM, Redd SC. Involuntary smoking and asthma severity in children: data from the Third National Health and Nutrition Examination Survey. *Chest*. 2002;122(2):409–415.
5. Chilmonczyk BA, Salmun LM, Megathlin KN, et al. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. *N Engl J Med*. 1993;328(23):1665–1669.
6. Evans D, Levison MJ, Feldman CH, et al. The impact of passive smoking on emergency room visits of urban children with asthma. *Am Rev Respir Dis*. 1987;135(3):567–572.
7. Sippel JM, Pedula KL, Vollmer WM, Buist AS, Osborne ML. Associations of smoking with hospital-based care and quality of life in patients with obstructive airway disease. *Chest*. 1999;115(3):691–696.
8. LeSon S, Gershwin ME. Risk factors for asthmatic patients requiring intubation. I. Observations in children. *J Asthma*. 1995;32(4):285–294.
9. Gerald LB, Gerald JK, Gibson L, Patel K, Zhang S, McClure LA. Changes in environmental tobacco smoke exposure and asthma morbidity among urban school children. *Chest*. 2009;135(4):911–916.
10. Wilson KMGS, Wesgate SC, Best D, Blumkin AK, Klein JD. Admission screening for secondhand tobacco smoke exposure. *Hosp Pediatr*. 2012;2(1):26–33.
11. National Asthma Education and Prevention Program. Expert Panel Report 3 (EPR-3): guidelines for the diagnosis and management of asthma—summary report 2007. *J Allergy Clin Immunol*. 2007;120(5 suppl):S94–S138.
12. van den Bosch GE, Merkus PJ, Buysse CM, et al. Risk factors for pediatric intensive care admission in children with acute asthma. *Respir Care*. 2012;57(9):1391–1397.

13. Lyell PJ, Villanueva E, Burton D, Freezer NJ, Bardin PG. Risk factors for intensive care in children with acute asthma. *Respirology*. 2005;10(4):436–441.
14. Berry JG, Hall DE, Kuo DZ, et al. Hospital utilization and characteristics of patients experiencing recurrent readmissions within children's hospitals. *JAMA*. 2011;305(7):682–690.
15. Miller MK, Lee JH, Miller DP, Wenzel SE; TENOR Study Group. Recent asthma exacerbations: a key predictor of future exacerbations. *Respir Med*. 2007;101(3):481–489.
16. Winickoff JP, Hillis VJ, Palfrey JS, Perrin JM, Rigotti NA. A smoking cessation intervention for parents of children who are hospitalized for respiratory illness: the stop tobacco outreach program. *Pediatrics*. 2003;111(1):140–145.
17. Ralston S, Roohi M. A randomized, controlled trial of smoking cessation counseling provided during child hospitalization for respiratory illness. *Pediatr Pulmonol*. 2008;43(6):561–566.
18. Howrylak JA, Spanier AJ, Huang B, et al. Cotinine in children admitted for asthma and readmission. *Pediatrics*. 2014;133(2). Available at: www.pediatrics.org/cgi/content/full/133/2/e355.

# Is Secondhand Smoke Exposure Associated With Increased Exacerbation Severity Among Children Hospitalized for Asthma?

Annie L. Andrews, Nils Shirley, Elizabeth Ojukwu, Michelle Robinson, Michelle Torok and Karen M. Wilson

*Hospital Pediatrics* 2015;5;249
DOI: 10.1542/hpeds.2014-0128

# Is Secondhand Smoke Exposure Associated With Increased Exacerbation Severity Among Children Hospitalized for Asthma?

Annie L. Andrews, Nils Shirley, Elizabeth Ojukwu, Michelle Robinson, Michelle Torok and Karen M. Wilson

*Hospital Pediatrics* 2015;5;249
DOI: 10.1542/hpeds.2014-0128