Published in final edited form as:

Int Forum Allergy Rhinol. 2015 June ; 5(6): 471–476. doi:10.1002/alr.21444.

Allergic sensitization, rhinitis, and tobacco smoke exposure in U.S. children and adolescents

Josef Shargorodsky, MD, MPH¹, Esther Garcia-Esquinas, MD, MPH, PhD^{2,3}, Ana Navas-Acien, MD, PhD^{4,5}, and Sandra Y. Lin, MD¹

¹Department of Otolaryngology–Head and Neck Surgery, Johns Hopkins University School of Medicine, Baltimore, MD

²Department of Preventive Medicine and Public Health, School of Medicine, Autonoma University, Madrid, Spain

³Ciber of Epidemiology and Public Health (CIBERESP), Madrid, Spain

⁴Department of Environmental Health Sciences, Johns Hopkins University Bloomberg School of Public Health, Baltimore, MD

⁵Welch Center for Prevention, Epidemiology, and Clinical Research, Johns Hopkins Medical Institutions, Baltimore, MD

Abstract

Background—Childhood tobacco exposure has been linked with sinonasal pathology, and may be associated with allergic sensitization. This study evaluates the association between exposure to active smoking or secondhand smoke (SHS) and the prevalence of rhinitis and allergic sensitization in the U.S. pediatric population.

Methods—Cross-sectional study in 2714 children and adolescents aged 6 to 19 in the National Health and Nutrition Examination Survey (NHANES), 2005–2006. Active smoking was defined as self-reported smoking or serum cotinine concentrations >10 ng/mL. SHS was defined as nonactive smokers who reported living with 1 smokers or had serum cotinine 0.011 ng/mL. Self-reported rhinitis was based on symptoms during the past 12 months, and allergen sensitization was defined as a positive response to any of the 19 specific immunoglobulin E (IgE) antigens tested.

Results—About half of the population (54%) had detectable levels of IgE specific to at least 1 of the tested allergens, and 25% reported a history of rhinitis. After multivariate adjustment, an increased prevalence rate ratio (PRR) of self-reported rhinitis was seen in individuals in the highest cotinine tertile among active smokers (PRR, 1.73; 95% confidence interval [CI], 1.23 to 2.43), with a significant trend between increasing cotinine levels in individuals exposed to either secondhand smoke or active smoking ($p = 0.05$ for both analyses). Significantly less food allergen

Correspondence to: Josef Shargorodsky, MD, MPH, Department of Otolaryngology–Head and Neck Surgery, Johns Hopkins University School of Medicine, 601 North Caroline Street, 6th Floor, Baltimore, MD 21287-0910; jshargo1@jhmi.edu.

Potential conflict of interest: None provided.

sensitization was observed in participants in the highest cotinine tertile of secondhand smoke (PRR, 0.61; 95% CI, 0.43 to 0.85).

Conclusion—Tobacco smoke exposure was associated with increased prevalence of rhinitis symptoms, but decreased prevalence of allergic sensitization. The results highlight the complex relationship between tobacco exposure and sinonasal pathology.

Keywords

allergy; rhinitis; tobacco; smoking; children

Allergic sensitization is a predisposing factor for multiple pediatric respiratory conditions. These conditions, including asthma and allergic rhinitis among others, can have profound consequences on a child's quality of life, and incur a tremendous economic burden. In the United States, the total direct annual cost of allergic rhinitis alone has been shown to be as high as \$3.4 billion dollars.¹ The associated symptoms are very common, with symptoms of rhinitis having a reported annual incidence as high as over 30% in the U.S. pediatric population.² Despite the scope of the problem, few environmental risk factors have been identified for allergic sensitization, or specifically for allergic rhinitis.

Childhood tobacco exposure is common, and has been linked with an increased risk of multiple upper respiratory conditions. Past data on the relations between tobacco smoke exposure and pediatric allergic sensitization, however, have been conflicting. Significant associations have been suggested between tobacco exposure and allergic rhinitis.³ On the other hand, multiple studies have shown immunoglobulin E (IgE)-mediated allergic sensitization to be either not significantly associated,⁴ or inversely associated with tobacco exposure.⁵ This suggests that tobacco exposure may predispose children to symptoms of rhinitis, but this may or may not be associated with an increased predisposition to allergic sensitization.

Despite the high incidence of both childhood tobacco exposure and upper respiratory conditions, there is still no clear understanding of the relation between tobacco exposure and allergic sensitization or rhinitis. Large-scale pediatric population studies evaluating the associations between smoking exposure and these 2 conditions are lacking in the literature. Therefore, this study aimed to evaluate the association between exposure to active smoking or secondhand smoke (SHS) and the prevalence of rhinitis and allergic sensitization in the U.S. pediatric population.

Subjects and methods

Between 2005 and 2006, the National Health and Nutrition Examination Survey (NHANES) examined a nationally representative U.S. sample by using a complex multistage sample design. The NHANES protocol was reviewed and approved by the National Center for Health Statistics Institutional Review Board. For participants aged <18 years, informed consent was provided by the participants and their guardians. The unweighted response rate for children 6 to 19 years during the NHANES 2005–2006 was 84.7%.⁶

Study population

For this study, we selected individuals 6 to 19 years of age who participated in the NHANES 2005–2006 (n = 3433). We then excluded participants with missing values in serum cotinine (n = 555), allergic sensitization (n = 43), or rhinitis (n = 2), as well as those without information on sex, age, ethnicity, parental education level, or body mass index (BMI) (n = 119). Participants included in the analysis were similar with respect to the main sociodemographic variables when compared with the original sample of children (data not shown).

Exposure assessment

Serum cotinine was measured by an isotope dilution, high-performance liquid chromatography/atmospheric pressure chemical ionization tandem mass spectrometry (Centers for Disease Control and Prevention [CDC]). The limit of detection (LOD) for serum cotinine was 0.015 ng/mL. Serum cotinine concentrations below the LOD were replaced by the LOD divided by the square root of 2 (according to the CDC protocol for NHANES⁷).

Tobacco use and exposure to SHS were assessed by using self-reported information and serum cotinine. Participants reporting having smoked at least 1 day or 1 cigarette in the last month, as well as those who had serum cotinine concentrations over 10 ng/mL were classified as active smokers (n = 330). Nonactive smokers who reported living with at least 1 person who smoked or who had cotinine levels 0.011 ng/mL but 10 ng/mL were considered exposed to SHS (n = 1897). Finally, participants with serum cotinine levels <0.011 ng/mL, not living with a smoker, and not smoking in the last month were classified as unexposed to tobacco (n = 487). Participants exposed to SHS or active smoking were divided into 3 categories according to serum cotinine tertiles.

Outcome assessment

NHANES 2005–2006 collected information on allergic symptoms and sensitization. Serum samples were analyzed for 19 allergen specific IgEs (*Dermatophagoides farinae*, *Dermatophagoides pteronyssinus*, cat, dog, cockroach, *Alternaria alternata*, peanut, egg, cow's milk, ragweed, rye grass, Bermuda grass, oak, birch, shrimp, *Aspergillus fumigatus*, Russian thistle, mouse, and rat) using the Pharmacia Diagnostics ImmunoCAP 1000 System (Kalamazoo, MI). The lower limit of detection for each of the allergen-specific IgE antibody tests was 0.35 kU/L. Allergen sensitization was defined as a positive response if the level of any of the 19 specific IgE antigens was above the lower limit of detection (0.35 kU/L). Additionally inhaled allergen sensitization was defined as a positive response to *Dermatophagoides farinae*, *Dermatophagoides pteronyssinus*, cat, dog, cockroach, *Alternaria alternata*, ragweed, rye grass, Bermuda grass, oak, birch, *Aspergillus fumigatus*, Russian thistle, mouse, and rat; food allergen sensitization was defined as a positive response to peanut, egg, cow's milk, or shrimp specific IgEs. Self-reported rhinitis was defined an affirmative reply to the question “during the last 12 months have you had a problem with sneezing, or a runny, or blocked nose when you did not have a cold or the flu?”

Adjustment variables

Questionnaire information included gender, age, education of the household reference person (less than high school, high school, and more than high school), race/ethnicity (non-Hispanic white, non-Hispanic black, Mexican American, other) and specific BMI percentiles (84, 85 to 94, 95) for age and gender based on the CDC's growth charts.

Statistics

Statistical analyses were performed in STATA version 11.2 statistical software (Stata Corp, College Station, TX) by using the survey (svy) command to account for the complex sampling design and weights in the NHANES.

The prevalence of allergic sensitization, inhalant allergen sensitization, food allergen sensitization, and rhinitis was calculated overall and by sex, age, ethnicity, parental education, and BMI percentiles. We also estimated adjusted prevalence rate ratios (PPRs) and their 95% confidence intervals (CIs) to assess the association between tobacco smoke exposure and the studied outcome variables using Poisson regression models. The relation between tobacco smoke exposure and self-reported rhinitis, independent of allergic sensitization, was further explored by conducting a separate analysis of only the individuals with no allergic sensitization.

Results

A total of 2714 individuals were included in the study. As shown in Table 1, approximately 54% of participants (n = 1474) were sensitized to at least 1 allergen and 25% (n = 685) self-reported having rhinitis. Male, younger, and black children were more likely to have allergic sensitization. Older children, whites, and children whose parents had attended high school or above showed a higher prevalence of rhinitis. Around 12% and 70% were exposed to active smoking or SHS, respectively. Most of the children with a history of active smoking (82%) were in the oldest age group.

Results for the association between allergic sensitization, self-reported rhinitis, and tobacco smoke exposure are shown in Table 2. Three models are shown: a univariate model (model 1), a multivariate model adjusted for age and gender (model 2), and a multivariate model adjusted for age, gender, ethnicity, parental education, and BMI (model 3). After multivariate adjustment for age, gender, ethnicity, parental education, and BMI, tobacco smoke exposure was only associated with food allergen sensitization and with self-reported rhinitis. While a decreased prevalence of food allergen sensitization was observed in participants in the highest cotinine tertile of SHS (PRR, 0.61; 95% CI, 0.43 to 0.85), an increased frequency of self-reported rhinitis was seen in individuals in the highest cotinine tertile of active smoking (PRR, 1.73; 95% CI, 1.23 to 2.43). A significant trend was apparent between increasing cotinine levels in individuals exposed to either SHS or active smoking, and prevalence of self-reported rhinitis ($p = 0.05$ for both analyses).

In order to further evaluate the relation between tobacco smoke exposure and rhinitis, independent of allergic sensitization, an analysis of individuals with no allergic sensitization was performed (Table 3). A stronger association was observed between rhinitis and the

highest cotinine tertile in the active smoking group (PRR, 2.16) than in analyses including the allergic-sensitized individuals. There was also a significant trend toward increasing rhinitis prevalence with increasing cotinine tertiles in individuals exposed to SHS ($p = 0.02$).

Discussion

This study evaluated the association between exposure to active smoking or SHS and the prevalence of rhinitis and allergic sensitization in the U.S. pediatric population. The prevalence of rhinitis in our cohort was consistent with previous reports using NHANES data^{8,9} as well as with other U.S. nationwide surveys.¹⁰ The assessment of tobacco smoke exposure in this study was highly sensitive, assigning tobacco exposure to individuals with even very low levels of serum cotinine. This method of assessing tobacco exposure in the NHANES dataset has been described, and the prevalence of exposure in this study was consistent with the prior report.¹¹ Children identified as either active smokers or exposed to SHS appeared to have a greater prevalence of rhinitis symptoms with increasing levels of serum cotinine. This relationship did not appear to be due to allergic sensitization, because tobacco smoke exposure was not significantly associated with allergic sensitization, except to food allergens. Sensitization to food allergens was inversely related to smoking exposure, as the prevalence of sensitization decreased with increasing serum cotinine levels. In addition, an analysis of nonsensitized individuals demonstrated even stronger associations between tobacco smoke exposure and rhinitis.

Associations between tobacco smoke exposure and multiple respiratory diseases have been documented in the literature; a study of 200 individuals in 2011 demonstrated both past and current SHS exposure to be a significant risk factor for allergic rhinitis.¹² Likewise, both active smoking and SHS exposure have shown an association with increased prevalence of chronic rhinosinusitis.¹³ In children, significant associations with tobacco smoke exposure have been reported with allergic rhinitis,¹⁴ asthma,¹⁵ bronchitis,¹⁶ and chronic cough.¹⁷ In addition, increasing cotinine levels in children have been shown to be associated with worse pulmonary function test results in a dose-dependent manner.¹⁸ Smoking exposure has been shown to modify the physiology of the normal respiratory tract, with demonstrated thickening of lower airway walls, impairment of mucociliary clearance, and alteration of airway immune function.¹⁹ The specific mechanism for the relation between rhinitis and tobacco smoke exposure, however, has not been proven.

Although there is a concern that allergic sensitization is a potential mechanism for development of rhinitis in children exposed to tobacco smoke, our study showed that the association between rhinitis prevalence and tobacco exposure may be independent of allergic sensitization. This is consistent with other recent findings. A cross-sectional study of 18,087 individuals in Sweden found a higher prevalence of allergic rhinitis associated with nonsmokers than smokers, but a higher prevalence of chronic sinusitis and nasal congestion associated with a positive smoking history.²⁰ The results suggest that the mechanisms linking smoking and IgE-mediated allergic sensitization may be distinct from those linking smoking with rhinitis.

The relationship between allergic sensitization and tobacco smoke exposure is complex. Although there is evidence of significant associations between atopic conditions and tobacco smoke exposure, evidence has also been demonstrated for a potentially inverse relationship between smoking exposure and mean IgE measurements in children.⁵ In addition, a lower absolute eosinophil count has been observed in active smokers compared to never smokers.²¹ Results from work looking at chronic airway inflammation have also suggested distinct eosinophilic and non-eosinophilic (neutrophilic predominant) phenotypes of inflammation, with potentially distinct clinical disease courses and responses to treatment.²² Epidemiologic studies have also shown that smoking may contribute to the development of nonallergic rhinitis^{23,24} through neurogenic inflammation induced by cigarette smoke.²⁵ Neurogenic inflammation resulting from environmental exposures has been well described in the literature and represents an inflammatory mechanism distinct from that of allergic sensitization.²⁶ The nonallergic etiology of nasal inflammation is further supported by multiple studies showing a lack of association between aeroallergen sensitization and tobacco smoke exposure.^{4,27} The physiologic mechanism of the association identified in our study may also be the result of the previously documented immunosuppressive effects of tobacco smoke. In this sense, tobacco smoke has been shown to inhibit the expression of proinflammatory cytokines,²⁸ reduce serum levels of immunoglobulins, and inhibit T cell activation.²⁹ In fact, this inhibition of proinflammatory mediators has been hypothesized to cause an increase in the incidence of pulmonary infections in smokers.³⁰ Tobacco smoke has been demonstrated to inhibit multiple inflammatory mediators, leading to a variety of infections.^{31,32} These mechanisms may explain the difference in the associations with smoking exposure between self-reported rhinitis and allergic sensitization in our study.

The strengths and limitations of our study should be considered. Data from NHANES are comprehensive and nationally representative, drawing from a large and diverse sample of participants. The NHANES tobacco smoke exposure measures have been shown to be objective and reliable in past studies.¹¹ The study, however, is cross-sectional, and therefore causality with respect to exposure variables cannot be determined. Rhinitis was self-reported, and so may be prone to recall bias. However, we expect that recall bias would be similar in participants exposed and those unexposed to tobacco smoke. Although multivariate adjusted models were used in the analyses, there is also the possibility of residual confounding by unmeasured factors. This may be especially problematic in children exposed to environmental tobacco smoke because this population has also been shown to be at an increased risk for other potentially harmful exposures.^{33,34} Reverse causation may also play a role, with the parents of children with diagnosed allergic sensitization or respiratory conditions potentially protecting the children from further tobacco exposure. In our study population, however, the prevalence of smoking exposure was high, and with a wide distribution of cotinine measurements, making this phenomenon less likely to affect the study outcomes.

Conclusion

This study demonstrated a significantly higher prevalence of rhinitis symptoms in children with tobacco smoke exposure, and this was independent of allergic sensitization. There was no significant association with inhaled allergen sensitization, and a decreased prevalence of

food allergen sensitization in children exposed to tobacco smoke. The results of this study highlight the complex relationship between tobacco smoke exposure and sinonasal pathology, and suggest that the effect of smoking exposure on rhinitis symptoms may have a mechanism distinct from allergic sensitization. Regardless of the mechanism, however, the observed associations between rhinitis and tobacco smoke exposure provide further evidence for the importance of minimizing tobacco exposure in the pediatric population.

References

1. Meltzer EO, Bukstein DA. The economic impact of allergic rhinitis and current guidelines for treatment. *Ann Allergy Asthma Immunol.* 2011; 106:S12–S16. [PubMed: 21277528]
2. Hoppin JA, Jaramillo R, Salo P, Sandler DP, London SJ, Zeldin DC. Questionnaire predictors of atopy in a US population sample: findings from the National Health and Nutrition Examination Survey, 2005–2006. *Am J Epidemiol.* 2011; 173:544–552. [PubMed: 21273397]
3. Saulyte J, Regueira C, Montes-Martinez A, Khudyakov P, Takkouche B. Active or passive exposure to tobacco smoking and allergic rhinitis, allergic dermatitis, and food allergy in adults and children: a systematic review and meta-analysis. *PLoS Med.* 2014; 11:e1001611. [PubMed: 24618794]
4. Ciacchio CE, DiDonna AC, Kennedy K, Barnes CS, Portnoy JM, Rosenwasser LJ. Association of tobacco smoke exposure and atopic sensitization. *Ann Allergy Asthma Immunol.* 2013; 111:387–390. [PubMed: 24125146]
5. Bottema RWB, Reijmerink NE, Kerkhof M, et al. Interleukin 13, CD14, pet and tobacco smoke influence atopy in three Dutch cohorts: the allergenic study. *Eur Respir J.* 2008; 32:593–602. [PubMed: 18417506]
6. Centers for Disease Control and Prevention. [Accessed October 24, 2014] National Health and Nutrition Examination Survey (NHANES) response rates and CPS totals. http://www.cdc.gov/nchs/nhanes/response_rates_CPS.htm
7. Centers for Disease Control and Prevention. [Accessed October 24, 2014] National Health and Nutrition Examination Survey. http://www.cdc.gov/nchs/nhanes/about_nhanes.htm
8. Hoppin JA, Jaramillo R, London SJ, et al. Phthalate exposure and allergy in the U.S. population: results from NHANES 2005–2006. *Environ Health Perspect.* 2013; 121:1129–1134. [PubMed: 23799650]
9. Salo PM, Calatroni A, Gergen PJ, et al. Allergy-related outcomes in relation to serum IgE: results from the National Health and Nutrition Examination Survey 2005–2006. *J Allergy Clin Immunol.* 2011; 127:1226.e7–1235.e7. [PubMed: 21320720]
10. Nathan RA, Meltzer EO, Derebery J, et al. The prevalence of nasal symptoms attributed to allergies in the United States: findings from the burden of rhinitis in an America survey. *Allergy Asthma Proc.* 2008; 29:600–608. [PubMed: 19173786]
11. Garcia-Esquinas E, Loeffler LF, Weaver VM, Fadrowski JJ, Navas-Acien A. Kidney function and tobacco smoke exposure in US adolescents. *Pediatrics.* 2013; 131:e1415–e1423. [PubMed: 23569089]
12. Lin SY, Reh DD, Clipp S, Irani L, Navas-Acien A. Allergic rhinitis and secondhand tobacco smoke: a population-based study. *Am J Rhinol Allergy.* 2011; 25:e66–e71. [PubMed: 21679502]
13. Reh DD, Higgins TS, Smith TL. Impact of tobacco smoke on chronic rhinosinusitis: a review of the literature. *Int Forum Allergy Rhinol.* 2012; 2:362–369. [PubMed: 22696460]
14. Hur K, Liang J, Lin SY. The role of secondhand smoke in allergic rhinitis: a systematic review. *Int Forum Allergy Rhinol.* 2014; 4:110–116. [PubMed: 24493468]
15. Quinto, KB.; Kit, BK.; Lukacs, SL.; Akinbami, LJ. NCHS Data Brief, no 126. Hyattsville, MD: National Center for Health Statistics; 2013. Environmental tobacco smoke exposure in children aged 3–19 years with and without asthma in the United States, 1999–2010. <http://www.cdc.gov/nchs/data/databriefs/db126.pdf> [Accessed October 24, 2014]
16. Tsai C-H, Huang J-H, Hwang B-F, Lee YL. Household environmental tobacco smoke and risks of asthma, wheeze and bronchitic symptoms among children in Taiwan. *Respir Res.* 2010; 11:11. [PubMed: 20113468]

17. Singh D, Arora V, Sobti PC. Chronic/recurrent cough in rural children in Ludhiana, Punjab. *Indian Pediatr.* 2002; 39:23–29. [PubMed: 11805350]
18. Valsamis C, Krishnan S, Dozor A. The effects of low-level environmental smoke exposure on pulmonary function tests in preschool children with asthma. *J Asthma.* 2014; 51:685–690. [PubMed: 24575853]
19. Hogg JC. Pathophysiology of airflow limitation in chronic obstructive pulmonary disease. *Lancet.* 2004; 364:709–721. [PubMed: 15325838]
20. Eriksson J, Ekerljung L, Pullerits T, et al. Prevalence of chronic nasal symptoms in West Sweden: risk factors and relation to self-reported allergic rhinitis and lower respiratory symptoms. *Int Arch Allergy Immunol.* 2011; 154:155–163. [PubMed: 20733324]
21. Berania I, Endam L, Filali-Mouhim A, et al. Active smoking status in chronic rhinosinusitis is associated with higher serum markers of inflammation and lower serum eosinophilia. *Int Forum Allergy Rhinol.* 2014; 4:347–352. [PubMed: 24431239]
22. Gibson PG, Simpson JL, Saltos N. Heterogeneity of airway inflammation in persistent asthma: evidence of neutrophilic inflammation and increased sputum interleukin-8. *Chest.* 2001; 119:1329–1336. [PubMed: 11348936]
23. Montnemery P, Svensson C, Adelroth E, et al. Prevalence of nasal symptoms and their relation to self-reported asthma and chronic bronchitis/emphysema. *Eur Respir J.* 2001; 17:596–603. [PubMed: 11401051]
24. Olsson P, Berglund N, Bellander T, Stjarne P. Prevalence of self-reported allergic and non-allergic rhinitis symptoms in Stockholm: relation to age, gender, olfactory sense and smoking. *Acta Otolaryngologica.* 2003; 123:75–80.
25. Lundblad L, Lundberg JM. Capsaicin sensitive sensory neurons mediate the response to nasal irritation induced by the vapour phase of cigarette smoke. *Toxicology.* 1984; 33:1–7. [PubMed: 6495343]
26. Meggs WJ. Neurogenic inflammation and sensitivity to environmental chemicals. *Environ Health Perspect.* 1993; 101:234–238. [PubMed: 8404760]
27. Kulig M, Luck W, Lau S, et al. Effect of pre- and postnatal tobacco smoke exposure on specific sensitization to food and inhalant allergens during the first 3 years of life. Multicenter Allergy Study Group, Germany. *Allergy.* 1999; 54:220–228. [PubMed: 10321557]
28. Ouyang Y, Virasch N, Hao P, et al. Suppression of human IL-1beta, IL-2, IFN-gamma, and TNF-alpha production by cigarette smoke extracts. *J Allergy Clin Immunol.* 2000; 106:280–287. [PubMed: 10932071]
29. Sopori M. Effects of cigarette smoke on the immune system. *Nat Rev Immunol.* 2002; 2:372–377. [PubMed: 12033743]
30. Chen H, Cowan MJ, Hasday JD, Vogel SN, Medvedev AE. Tobacco smoking inhibits expression of proinflammatory cytokines and activation of IL-1R-associated kinase, p38, and NF-kappaB in alveolar macrophages stimulated with TLR2 and TLR4 agonists. *J Immunol.* 2007; 179:6097–6106. [PubMed: 17947684]
31. Ryder MI, Saghizadeh M, Ding Y, Nguyen N, Soskolne A. Effects of tobacco smoke on the secretion of interleukin-1beta, tumor necrosis factor-alpha, and transforming growth factor-beta from peripheral blood mononuclear cells. *Oral Microbiol Immunol.* 2002; 17:331–336. [PubMed: 12485323]
32. Kum-Nji P, Meloy L, Herrod HG. Environmental tobacco smoke exposure: prevalence and mechanisms of causation of infections in children. *Pediatrics.* 2006; 117:1745–1754. [PubMed: 16651333]
33. Moore K, Borland R, Yong H- et al. Support for tobacco control interventions: do country of origin and socioeconomic status make a difference? *Int J Public Health.* 2012; 57:777–786. [PubMed: 22714136]
34. Holtby S, Zahnd E, Grant D, Park R. Children's exposure to secondhand smoke: nearly one million affected in California. Policy Brief UCLA Cent Health Policy Res. 2011:1–8. <http://healthpolicy.ucla.edu/publications/Documents/PDF/SmokePBREVISED11-2-11.pdf>.

TABLE 1

AS, self-reported rhinitis, and tobacco smoke exposure status by participant characteristics in U.S. children 6 to 19 years of age

Demographic factors	AS: any (n = 1474)	AS: inhaled (n = 1379)	AS: food (n = 672)	Rhinitis (n = 685)	Smoking unexposed (n = 487)	SHS (n = 1897)	Active smoking (n = 330)
Gender							
Male	55.0	55.4	58.0	49.5	50.0	50.8	57.6
Female	45.0	44.6	42.0	50.5	50.0	49.2	42.4
Age (years)							
6–11	38.0	35.6	41.8	35.6	43.6	44.7	0.7
12–15	30.7	31.8	32.0	30.1	37.0	32.2	17.3
16–19	31.3	32.6	26.2	34.3	19.4	23.1	82.0
Ethnicity							
White	56.6	56.7	53.3	65.8	61.4	60.1	69.5
Black	19.5	19.9	22.2	12.4	6.0	17.3	11.1
Mexican	12.8	12.7	12.1	8.4	19.5	11.3	8.0
Other	11.1	10.7	12.4	13.4	13.1	11.3	11.4
Education							
<HS	19.0	19.0	20.6	12.2	12.8	19.3	24.8
HS	25.5	25.4	23.0	25.3	13.8	28.0	33.6
>HS	55.5	55.6	56.4	62.5	73.4	52.7	41.6
BMI							
Normal	65.3	65.2	62.9	69.9	72.6	65.6	65.0
Overweight	15.8	15.5	15.9	13.3	12.5	15.9	14.1
Obese	18.9	19.3	21.2	17.1	14.9	18.5	20.9

* Values are weighted percents. Total number of children, n = 2714.

AS = allergic sensitization; BMI = body mass index; HS = high school; SHS = second-hand smoke.

TABLE 2

Prevalence ratio estimates and 95% confidence intervals for the association between allergic sensitization, rhinitis and tobacco smoke exposure by serum cotinine tertiles*

	n	Any allergic sensitization			Inhaled allergen sensitization			Food allergen sensitization			Rhinitis self-report		
		Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Unexposed	487	1	1	1	1	1	1	1	1	1	1	1	1
SHS													
0.011–0.038	611	0.87 (0.71–1.07)	0.86 (0.71–1.06)	0.85 (0.70–1.05)	0.84 (0.64–1.11)	0.83 (0.63–1.09)	0.82 (0.62–1.07)	0.93 (0.67–1.29)	0.93 (0.67–1.29)	0.90 (0.65–1.25)	0.85 (0.54–1.33)	0.85 (0.54–1.33)	0.87 (0.58–1.31)
0.039–0.182	622	1.06 (0.86–1.30)	1.05 (0.86–1.29)	1.02 (0.83–1.24)	1.08 (0.85–1.40)	1.07 (0.84–1.36)	1.03 (0.82–1.30)	1.07 (0.81–1.42)	1.07 (0.81–1.41)	0.98 (0.74–1.31)	1.02 (0.72–1.45)	1.02 (0.72–1.45)	1.10 (0.82–1.45)
0.183–10	664	0.91 (0.75–1.09)	0.91 (0.76–1.09)	0.85 (0.71–1.02)	0.88 (0.70–1.10)	0.88 (0.71–1.10)	0.82 (0.67–1.00)	0.71 (0.53–0.96)	0.72 (0.54–0.96)	0.61 (0.43–0.85)	0.97 (0.69–1.36)	0.97 (0.69–1.36)	1.12 (0.85–1.49)
p trend		0.63	0.5	0.97	0.7	0.53	0.98	0.11	0.13	0.03	0.2	0.2	0.05
Active smoking													
0.011–25.9	130	1.13 (0.87–1.47)	1.06 (0.83–1.34)	1.05 (0.83–1.34)	1.19 (0.87–1.63)	1.03 (0.76–1.40)	1.02 (0.76–1.38)	0.84 (0.48–1.48)	0.90 (0.48–1.66)	0.87 (0.46–1.62)	1.05 (0.64–1.70)	1.01 (0.64–1.59)	1.09 (0.69–1.72)
26–120	108	1.11 (0.83–1.50)	1.04 (0.75–1.44)	1.06 (0.76–1.46)	1.14 (0.82–1.58)	0.99 (0.76–1.40)	1.00 (0.71–1.42)	0.98 (0.56–1.74)	1.05 (0.53–2.10)	1.03 (0.51–2.05)	1.15 (0.77–1.73)	1.10 (0.68–1.79)	1.24 (0.82–1.87)
121	92	0.99 (0.68–1.42)	0.94 (0.64–1.36)	0.96 (0.68–1.35)	1.02 (0.71–1.46)	0.89 (0.63–1.27)	0.91 (0.66–1.25)	0.82 (0.47–1.43)	0.90 (0.52–1.57)	0.86 (0.54–1.37)	1.56 (1.06–2.31)	1.49 (0.95–2.32)	1.73 (1.23–2.43)
p trend		0.55	0.59	0.66	0.47	0.5	0.57	0.96	0.98	0.99	0.1	0.11	0.05

* Values are prevalence ratio (95% confidence interval).

SHS = second-hand smoke.

TABLE 3

Prevalence ratio estimates and 95% confidence intervals for the association between rhinitis and tobacco smoke exposure status in nonsensitized children*

	n	Rhinitis self-report		
		Model 1	Model 2	Model 3
Unexposed	233	1	1	1
SHS				
0.011–0.038	300	0.89 (0.52–1.53)	0.91 (0.53–1.55)	0.92 (0.57–1.50)
0.039–0.182	265	0.91 (0.49–1.66)	0.92 (0.51–1.65)	1.00 (0.61–1.66)
0.183–10	306	1.25 (0.80–1.95)	1.29 (0.83–1.99)	1.56 (1.03–2.38)
<i>p</i> trend		0.06	0.07	0.02
Active smoking				
0.011–25.9	53	1.08 (0.44–2.64)	1.13 (0.48–2.66)	1.23 (0.56–2.70)
26–120	43	1.47 (0.74–2.90)	1.48 (0.63–2.47)	1.73 (0.85–2.54)
121	40	2.02 (1.08–3.76)	1.91 (0.90–4.06)	2.16 (1.00–4.68)
<i>p</i> trend		0.19	0.29	0.28

* Values are prevalence ratio (95% confidence interval).

SHS = second-hand smoke.