Secondhand Smoke as a Potential Cause of Chronic Rhinosinusitis

A Case-Control Study

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Objective: To assess the role of secondhand smoke (SHS) in the etiology of chronic rhinosinusitis (CRS).

Design: Matched case-control study. Associations between SHS and CRS were evaluated by conditional logistic regression odds ratios.

Setting: Henry Ford Health System, Detroit, Michigan.

Participants: A total of 306 nonsmoking patients diagnosed as having an incident case of CRS and 306 age-matched, sex-matched, and race/ethnicity–matched nonsmoking control patients.

Main Outcome Measures: Exposure to SHS for the 5 years before diagnosis of CRS (case patients) and before study entry (controls) for 4 primary sources: home, work, public places, and private social functions outside the home, such as parties, dinners, and weddings.

Results: Of controls and case patients, respectively, 28 (9.1%) and 41 (13.4%) had SHS exposure at home, 21 (6.9%) and 57 (18.6%) at work, 258 (84.3%) and 276 (90.2%) in public places, and 85 (27.8%) and 157 (51.3%) at private social functions. Adjusted for potential confounders (socioeconomic status and exposures to air pollution and chemicals or respiratory irritants from hobbies, work, or elsewhere), the odds ratios for CRS were 1.69 (95% confidence interval, 0.92-3.10) for SHS exposure at home, 2.81 (1.42-5.57) for exposure at work, 1.48 (0.88-2.49) for exposure in public places, and 2.60 (1.74-3.89) for exposure at private functions. A strong, independent dose-response relationship existed between CRS and the number of venues where SHS exposure occurred (odds ratio per 1 of 4 levels, 2.03; 95% confidence interval, 1.55-2.66). Approximately 40.0% of CRS appeared to be attributable to SHS.

Conclusions: Exposure to SHS is common and significantly independently associated with CRS. These findings have important clinical and public health implications.

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THE DETRIMENTAL HEALTH effects of smoking are well recognized,[1] and evidence of the adverse health effects of exposure to secondhand smoke (SHS) is accumulating.[2-6] Secondhand smoke contains more than 4000 substances, of which more than 50 are known or suspected carcinogens and many are strong irritants.[7] Several expert panel reviews of the health effects of SHS[5-9] have concluded that causal associations exist between SHS and sudden infant death syndrome, acute respiratory infections, middle ear disease, and asthma in children and coronary heart disease and lung and sinus cancers in adults. Evidence is suggestive but not conclusive regarding a causal relationship between SHS and many other diseases. Thus, SHS is a major public health problem because 60% of nonsmokers in the United States (126 million nonsmokers) are exposed to SHS.[6]

Most studies[6] of the associations between SHS exposure and respiratory disease in adults have investigated odor and irritation, respiratory symptoms, lung function, asthma, chronic obstructive pulmonary disease, and lung cancer. Few studies[10,11] have focused on chronic rhinosinusitis (CRS), although evidence suggests that such a relationship may exist. Chronic rhinosinusitis is a group of diseases with heterogeneous etiologies characterized by inflammation of the mucosa of the nose and paranasal sinuses lasting

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12 weeks or longer.[12] Factors associated with CRS include mucociliary impairment, allergy, bacterial or fungal infection, immunocompromised state, and environmental and genetic factors.[12] Chronic

rhinosinusitis is common in the United States, with an estimated prevalence of up to 16% in adults and accounting for approximately 22 million physician visits and more than 500 000 emergency department visits annually.[12,13]

Some studies[14-16] have found an association between SHS exposure and acute and chronic nasal and sinus symptoms. Acute respiratory inflammation characterized by nasal congestion, irritation, and increased rhinitis follows moderate SHS exposure in healthy adults.[17] The 2006 US Surgeon General's report[6] on the health consequences of involuntary exposure to tobacco smoke concluded that a causal relationship exists between SHS exposure and nasal irritation. Our study aimed to evaluate the association between SHS exposure and CRS.

METHODS

STUDY DESIGN

The study used a matched 1:1 case-control design. Case patients (n=306) were never smokers or former smokers who had adult-onset CRS. Case patients were identified through the Henry Ford Health System (HFHS) corporate database and had been diagnosed as having CRS and treated in the HFHS Department of Otolaryngology. Case patients were diagnosed as having chronic sinusitis (*International Classification of Diseases, Ninth Revision*[18] code 473) or nasal polyps (*International Classification of Diseases, Ninth Revision* code 471), which often accompany and are an indicator of CRS, and the diagnosis had to have been confirmed by computed tomography or nasal endoscopy. To ensure that we were evaluating incident vs prevalent cases, case patients had to have been free of CRS for at least 5 years before the index diagnosis. To the extent possible, the case definition followed the diagnostic criteria recommended by the Chronic Rhinosinusitis Task Force. All eligible incident case patients identified between January 1, 2000, and May 1, 2004, were considered for study enrollment.

Control patients were randomly selected from the HFHS corporate database and had been free of CRS for 5 years before study entry. Controls were matched to case patients by age (plus or minus 5 years), sex, and race/ethnicity. The HFHS is a large, comprehensive, nonprofit health care system that annually provides medical care to more than 500 000 people. In 1997, the HFHS patient population distribution in 10 age, 2 race/ethnicity, and the 2 sex categories differed from Metropolitan Detroit (1990 US Census) distributions by 5.3% or less in all strata. These observations suggest that the HFHS patient population is representative of the community it serves. To ensure comparability of data, study participants were required to be HFHS patients for 5 years or more before diagnosis (case patients) or study entry (controls). The HFHS institutional review board approved the study, and informed consent was obtained from all participants.

Study data were collected from administrative and medical records and by a structured questionnaire administered by telephone by trained HFHS interviewers. Study participants were masked to the hypothesis under investigation. We collected data on age, sex, race/ethnicity, socioeconomic status (educational attainment), diet, alcohol consumption, medical history, SHS exposures, and potential confounders (exposure to air pollution and chemicals or respiratory tract irritants at work, through hobbies, and from other sources). Comorbidity data collected by interview included information on diabetes mellitus, allergies, asthma, other respiratory diseases, oral health, headaches, and immune system diseases, such as human immunodeficiency virus, AIDS, autoimmune disease, or lupus.

The SHS exposure data for the 5 years before diagnosis with CRS (case patients) and before study entry (controls) were collected for 4 primary sources: home, work, public places, and private social functions outside the home, such as parties, dinners, and weddings. Private social functions were distinguished from public settings because they are often held in private dwellings with restricted access, and generally legislation has not attempted to restrict SHS exposure in such venues. We reasoned that capturing exposure data for the 5-year period before diagnosis would be meaningful for an inflammatory disease such as CRS. In this article, all exposures to SHS, air pollution, and environmental and occupational chemicals refers to the 5 years before diagnosis (case patients) or study entry (controls). The SHS data collection was based on a reliable approach designed by Brownson et al.[19]

STATISTICAL ANALYSIS

We evaluated distributions of study variables using contingency table analyses. The associations between CRS and SHS from each of the 4 primary sources of SHS exposure were evaluated by bivariate and multivariable analysis using conditional logistic regression and associated confidence intervals (CIs). Multivariable model adjustments, based on a priori reasoning, forced into models 5 potential confounders: (1) exposure to air pollution; exposure to chemicals or respiratory irritants (2) at work, (3) through hobbies, and (4) from nonwork and nonhobby sources; and (5) socioeconomic status, as estimated by individual educational level. For simplicity, confounders are presented in this report as dichotomous variables. Additional analyses were performed with confounders evaluated as 4-level indicator variables to assess to what extent the dichotomous results were biased by residual confounding. The results from the 2 approaches differed by less than 10% in all cases, and conclusions remained the same (data not shown).

The overall association between SHS and CRS was evaluated using conditional logistic regression analysis using an SHS score, which was created by summing the value of 1 for each source of SHS that was independently associated with CRS to which an individual was exposed. Interactions between SHS exposure and covariates in the final model were evaluated by including main effects terms and interaction terms in models and applying the likelihood ratio test to interaction terms.

To develop an understanding of the public health impact of SHS on CRS, we estimated the population-attributable risk for SHS in relation to CRS, using Levin's formula[20] for dichotomous measures of SHS exposure and Walter's formula[21] for multilevel exposures. These formulas are based on the frequencies of exposures in the population, which were estimated using exposure data from the control group, and relative risks, which were estimated by odds ratios (ORs). We estimated population-attributable risk (relative risk increase) using unadjusted estimates of ORs when confounding was absent.

Our prestudy estimated sample size had a power of 80.0% to detect an OR of 1.75, given an α error of .05 and 30.0% exposure in controls. Statistics were prepared with Stata/IC statistical software (StataCorp LP, College Station, Texas). All presented *P* values are 2-sided, and the α error was .05 for hypothesis testing.

RESULTS

STUDY POPULATION

Sociodemographic features of the study population and distributions of potential confounders are presented in

Table 1. Distribution of Sociodemographic and Non-SHS Variables by Case-Control Status and Associated Unadjusted Conditional Logistic Regression ORs

Variable	No. (%) Control Patients (n=306)	No. (%) Case Patients (n=306)	Unadjusted OR (95% CI)	*P* Value
Sociodemographic				
Age, mean (SD) [range], y	54.0 (14.7) [19-88]	53.6 (14.2) [19-86]	NA	.68[b]
Sex			NA	.99[c]
Male	135 (44.1)	135 (44.1)		
Female	171 (55.9)	171 (55.9)		
Race/ethnicity			NA	.60[c]
White	241 (78.8)	236 (77.1)		
Black	60 (19.6)	61 (19.9)		
Other	5 (1.6)	9 (2.9)		
Educational level[a]			0.91 (0.84-0.99)[a,d]	.03[d]
Eighth grade or less	3 (1.0)	3 (1.0)		
Some high school	8 (2.6)	19 (6.2)		
High school graduate	50 (16.4)	61 (20.0)		
Some technical school	13 (4.3)	8 (2.6)		
Some college	74 (24.3)	79 (25.9)		
College or university graduate	70 (23.0)	65 (21.3)		
Some postgraduate or professional study	16 (5.3)	19 (6.2)		
Completed postgraduate or professional degree	70 (23.0)	51 (16.7)		
Exposures to non-SHS risk factors				
Exposure to air pollution[a]				.04[c]
No	101 (35.1)	78 (27.0)	1 [Reference]	
Yes	187 (64.9)	211 (73.0)	1.59 (1.10-2.30)[e]	
Work exposures to irritating chemicals or fumes				<.001[c]
No	274 (89.5)	239 (78.1)	1 [Reference]	
Yes	32 (10.5)	67 (21.9)	2.59 (1.58-4.24)[e]	
Hobby exposures to irritating chemicals or fumes[a]				.002[c]
No	283 (92.5)	257 (84.5)	1 [Reference]	
Yes	23 (7.5)	47 (15.5)	2.92 (1.56-5.49)[e]	
Other (nonwork and nonhobby) exposures to irritating chemicals or fumes[a]				.01[c]
No	225 (73.5)	196 (61.3)	1 [Reference]	
Yes	81 (26.5)	109 (35.7)	1.52 (1.08-2.11)[e]	

Abbreviations: CI, confidence interval; NA, not applicable (because the study design was matched on age, sex, and race/ethnicity); OR, odds ratio; SHS, secondhand smoke.

[a] Numbers total to less than 306 for this variable because of missing data.

[b] Determined by *t* test.

[c] Determined by Fisher exact test.

[d] Odds ratio per 1 of 8 levels of change. Determined by correct nonparametric test of trend.

[e] Odds ratio for yes vs no.

Table 1. The study population had a mean age of 53.8 years, was 55.9% female, and was 77.9% white, 19.8% black, and 2.3% other or mixed race/ethnicity. Compared with controls, case patients had significantly lower educational levels and greater frequency of exposure to non-SHS risk factors (Table 1).

HOME EXPOSURE TO SHS

Of the study population, 28 controls (9.1%) and 41 case patients (13.4%) reported having been exposed to SHS at home. The unadjusted OR for exposure to SHS at home (some vs none) and CRS was 1.52 (95% CI, 0.92-2.52) and adjusted for potential confounders was 1.69 (0.92-3.10). The dose-response relationship between home SHS exposure and CRS was evaluated by grouping the total estimated hours of exposure to SHS in the home into 4 levels: no exposure and 3 similarly sized exposure groups (**Table 2**). In unadjusted analysis, this predictor, treated as an ordinal variable, had an unadjusted OR for a 1-level change of 1.26 (95% CI, 0.99-1.61). The adjusted OR did not demonstrate a clean dose response. The ORs for home SHS exposure and CRS with levels of exposure treated as indicator variables are presented in Table 2. A statistically nonsignificant increased risk is observed with all levels of exposure vs no exposure.

WORK EXPOSURE TO SHS

Overall, 21 controls (6.9%) and 57 case patients (18.6%) reported exposure to SHS at work. The unadjusted and adjusted ORs for work SHS exposure (some vs none) and CRS were 3.57 (95% CI, 1.97-6.46) and 2.81 (1.42-5.57), respectively. Individuals assessed their intensity of exposure to SHS at work in 4 levels (none, light, moderate, or heavy). Intensity of exposure to SHS at work, when assessed as an ordinal variable, had unadjusted and adjusted ORs for a 1-level change of 2.23 (95% CI, 1.52-3.26) and 2.27 (1.39-3.69), respectively. Intensity of exposure to SHS at work when evaluated as indicator

Table 2. Distribution of Selected Study Variables by Case-Control Status and Unadjusted and Adjusted Conditional Logistic Regression ORs[a]

Variable	No. (%) Control Patients (n=306)[b]	No. (%) Case Patients (n=306)[b]	Unadjusted Conditional Logistic OR (95% CI)	*P* Value	Adjusted[c] Conditional Logistic OR (95% CI)	*P* Value
Exposed to SHS at home						
No	278 (90.8)	265 (86.6)	1 [Reference]		1 [Reference]	
Yes	28 (9.1)	41 (13.4)	1.52 (0.92-2.52)[d]	.10	1.69 (0.92-3.10)[d]	.09
Home SHS exposure in past 5 y, mean (SD) [range], h	465 (2221) [0-27 375]	751 (2426) [0-18 250]				
Exposure in home in past 5 y						
L0 (0 h)	278 (90.8)	265 (86.6)	1.00 [Reference]		1 [Reference]	
L1 (>0-3000 h)	11 (3.6)	13 (4.2)	1.22 (0.55-2.74)[e]	.62	1.83 (0.68-4.99)[e]	.23
L2 (>3000-6000 h)	11 (3.6)	14 (4.6)	1.33 (0.56-3.16)[f]	.51	1.20 (0.46-3.17)[f]	.71
L3 (>6000 h)	6 (2.0)	14 (4.6)	2.36 (0.91-6.14)[g]	.08	2.55 (0.76-8.58)[g]	.13
Work SHS exposure ≥6 mo						
No	285 (93.1)	249 (81.4)	1.00 [Reference]		1 [Reference]	
Yes	21 (6.9)	57 (18.6)	3.57 (1.97-6.46)[d]	<.001	2.81 (1.42-5.57)[d]	.003
Self-evaluated SHS exposure at work						
L0 (none)	286 (93.5)	248 (81.0)	1.00 [Reference]		1.00 [Reference][h]	
L1 (light)	13 (4.2)	31 (10.1)	3.17 (1.53-6.56)[e]	.02	2.55 (1.18-5.52)[e]	.02
L2 (moderate)	6 (2.0)	15 (4.9)	3.44 (1.26-9.36)[f]	.02	2.65 (0.88-7.94)[f]	.08
L3 (heavy)	1 (0.3)	12 (3.9)	12.00 (1.56-92.29)[g]	.02	6.72 (0.84-54.05)[g]	.07
No. of public exposures to SHS per mo						
Mean (SD)	5.7 (6.3)	7.2 (9.4)				
Median (range)	4 (0-30)	4 (0-100)				
Public exposure to SHS						
No	48 (15.7)	30 (9.8)	1 [Reference]		1 [Reference]	
Yes	258 (84.3)	276 (90.2)	1.72 (1.05-2.82)[d]	.03	1.48 (0.88-2.49)[d]	.14
Public exposures to SHS times per mo						
L0 (0)	48 (15.7)	30 (9.8)	1 [Reference]		1 [Reference]	
L1 (1-4)	135 (44.1)	130 (42.5)	1.57 (0.93-2.64)[e]	.09	1.15 (0.63-2.09)[e]	.64
L2 (5-10)	81 (26.5)	87 (28.4)	1.76 (1.02-3.04)[f]	.04	1.17 (0.63-2.16)[f]	.62
L3 (11-100)	42 (13.7)	59 (19.3)	2.26 (1.22-4.21)[g]	.01	1.51 (0.75-3.05)[g]	.25
Public exposure to SHS, >10 times per mo						
No	263 (85.9)	248 (81.0)	1 [Reference]		1 [Reference]	
Yes	43 (14.1)	58 (19.0)	1.42 (0.92-2.17)[d]	.11	1.29 (0.80-2.09)[d]	.30
Self-estimated exposure to public SHS						
L0 (none)	48 (15.7)[i]	30 (9.9)[i]	1 [Reference]		1 [Reference]	
L1 (light)	171 (56.1)[i]	148 (49.0)[i]	1.40 (0.84-2.34)[e]	.20	1.05 (0.58-1.88)[e]	.87
L2 (moderate)	64 (21.0)[i]	98 (32.5)[i]	2.45 (1.40-4.31)[f]	.002	1.56 (0.83-2.93)[f]	.17
L3 (heavy)	22 (7.2)[i]	26 (8.6)[i]	1.99 (0.94-4.21)[g]	.07	1.10 (0.46-2.65)[g]	.83
No. of private exposures to SHS per mo, mean (SD)	0.6 (1.6)	1.3 (2.8)				
Mean (SD)	0.6 (1.6)	1.3 (2.8)				
Median (range)	0 (0-20)	1 (0-30)				
Private exposure to SHS						
No	221 (72.2)	149 (48.7)	1 [Reference]		1 [Reference]	
Yes	85 (27.8)	157 (51.3)	2.76 (1.93-3.94)[d]	<.001	2.60 (1.74-3.89)[d]	<.001
Private exposure to SHS, times per mo						
L0 (0)	221 (72.2)	149 (48.7)	1 [Reference]		1 [Reference]	
L1 (1)	42 (13.7)	72 (23.5)	2.52 (1.61-3.94)[e]	<.001	2.20 (1.33-3.63)[e]	.002
L2 (2)	19 (6.2)	45 (14.7)	3.53 (1.95-6.39)[f]	<.001	3.85 (1.98-7.49)[f]	<.001
L3 (>2)	24 (7.8)	40 (13.1)	2.57 (1.46-4.51)[g]	.001	2.44 (1.26-4.74)[g]	.008
Intensity of private exposure to SHS						
L0 (none)	221 (75.7)[i]	149 (49.2)[i]	1 [Reference]		1 [Reference]	
L1 (light)	44 (15.1)[i]	114 (37.6)[i]	3.72 (2.41-5.73)[e]	<.001	3.63 (2.22-5.93)[e]	<.001
L2 (moderate)	22 (7.5)[i]	32 (10.6)[i]	1.95 (1.03-3.67)[f]	.04	1.49 (0.73-3.06)[f]	.27
L3 (heavy)	5 (1.7)[i]	8 (2.6)[i]	2.91 (0.89-9.54)[g]	.08	4.09 (0.80-20.98)[g]	.09

Abbreviations: CI, confidence interval; L, level; OR, odds ratio; SHS, secondhand smoke.

[a] Percentages may not total 100 owing to rounding.

[b] Data are presented as number (percentage) of patients unless otherwise indicated.

[c] The model was adjusted for (1) exposure to air pollution; exposure to chemicals or respiratory irritants (2) at work, (3) through hobbies, and from (4) nonwork and nonhobby sources; and (5) socioeconomic status estimated by individual educational level.

[d] Odds ratio for yes vs no.

[e] Odds ratio for L1 vs L0.

[f] Odds ratio for L2 vs L0.

[g] Odds ratio for L3 vs L0.

[h] Inclusion of air pollution exposure in this model led to nonconvergence; thus, the variable "air pollution" was excluded from this model.

[i] Numbers total less than 306 for this variable because of missing data.

Table 3. Adjusted ORs for Chronic Rhinosinusitis From SHS Exposure From the 4 Primary Sources, With Exposure From Public Places Being Dichotomized Using 2 Different Cutpoints

Secondhand Smoke Exposure Source (Some vs None)	Univariate OR (95% CI)	*P* Value	OR (95% CI) Adjusted for Other SHS Sources	*P* Value	OR (95% CI) Adjusted for Other SHS Sources and Potential Confounders	*P* Value
			Cutpoint 1[a]			
Home	1.52 (0.92-2.52)	.10	1.48 (0.86-2.55)	.16	1.81 (0.95-3.43)	.07
Work	3.57 (1.97-6.46)	<.001	3.15 (1.71-5.82)	<.001	2.55 (1.25-5.21)	.01
Public	1.72 (1.05-2.82)	.03	1.29 (0.75-2.21)	.36	0.97 (0.53-1.78)	.92
Private	2.76 (1.93-3.94)	<.001	2.51 (1.73-3.65)	<.001	2.52 (1.67-3.82)	<.001
			Cutpoint 2[b]			
Home	1.52 (0.92-2.52)	.10	1.49 (0.86-2.56)	.15	1.85 (0.98-3.51)	.06
Work	3.57 (1.97-6.46)	<.001	3.15 (1.71-5.83)	<.001	2.57 (1.26-5.27)	.01
Public	1.42 (0.92-2.17)	.11	1.32 (0.83-2.11)	.23	1.27 (0.76-2.13)	.37
Private	2.76 (1.93-3.94)	<.001	2.57 (1.78-3.71)	<.001	2.49 (1.65-3.76)	<.001

Abbreviations: CI, confidence interval; OR, odds ratio; SHS, secondhand smoke.
[a] Public exposure was considered positive if greater than 0 exposures occurred per month.
[b] Public exposure was considered positive if greater than 10 exposures occurred per month.

variables demonstrated a dose-response relationship in unadjusted and adjusted models (Table 2).

EXPOSURE TO SHS IN PUBLIC PLACES

Among controls and case patients, respectively, 258 (84.3%) and 276 (90.2%) reported exposure to SHS in public places. Of exposed individuals, 531 (99.4%) reported where they received the greatest exposure to SHS: restaurants (62.0%), bars (14.9%), casinos (8.7%), bowling alleys (8.5%), and clubs (4.1%). The unadjusted and adjusted ORs for exposure to SHS in public places (some vs none) and CRS were 1.72 (95% CI, 1.05-2.82) and 1.48 (0.88-2.49), respectively. Dose of exposure to SHS in public places was estimated by the average number of exposures per month and by the self-reported estimate of intensity (none, light, moderate, or heavy). A significant dose response was only seen in the unadjusted association for the number of exposures per month (Table 2).

PRIVATE SOCIAL EXPOSURE TO SHS

Among controls and case patients, respectively, 85 (27.8%) and 157 (51.3%) reported attending private social functions where they were exposed to SHS. The unadjusted and adjusted ORs for SHS exposure at private social functions and CRS were 2.76 (95% CI, 1.93-3.94) and 2.60 (1.74-3.89), respectively. Average frequency of exposure to SHS at private functions per month and self-reported intensity of exposure at private functions (none, light, moderate, or heavy) were associated with CRS in unadjusted and adjusted analyses (Table 2), although no dose-response relationships were observed.

EXPOSURE TO SHS FROM THE 4 PRIMARY SOURCES

The ORs for CRS and each SHS source estimated in univariate and multivariable analyses are presented in **Table 3**. When the 4 sources of SHS were modeled as dichotomous variables (some vs no exposure) adjusted for each other and for the potential confounders, the ORs for CRS and SHS were 1.81 (95% CI, 0.95-3.43) for home exposure, 2.55 (1.25-5.21) for work exposure, 0.97 (0.53-1.78) for exposure in public places, and 2.52 (1.67-3.82) for private exposure. Each source of SHS, except for exposure in public places, was associated with increased odds of CRS. When exposure to SHS in public places was dichotomized at more than 10 exposures per month vs 0 to 10 exposures per month, the OR of CRS for public places was 1.27 (95% CI, 0.76-2.13) in the fully adjusted model (Table 3).

OVERALL SHS AND CRS ASSOCIATIONS

Table 4 presents the distribution and ORs for data when exposures to SHS at home, work, and private social functions are dichotomized at some vs none and exposure to SHS in public venues is dichotomized at more than 10 exposures per month vs 0 to 10 exposures. When these exposures were summed and treated as an ordinal variable (SHS score with 4 levels: 0, 1, 2, and 3-4 SHS exposures), the unadjusted and adjusted ORs for CRS per 1-level increase in the SHS score were 2.09 (95% CI, 1.65-2.63) and 2.03 (1.55-2.66), respectively. This relationship is described in the **Figure**. The adjusted SHS-CRS association was present in women (OR, 2.12; 95% CI, 1.45-3.12) and men (1.98; 1.32-2.98). When levels of SHS score were treated as indicator variables, they demonstrated a dose-response relationship in unadjusted and multivariable models (Table 4). Effect modification by asthma status or allergy status was not observed ($P = .52$ and $P = .98$ for the interaction term for asthma and allergies, respectively). In addition, study participants estimated their overall exposure to SHS from all sources by choosing 1 of 4 exposures (almost zero, light, moderate, or heavy). The unadjusted and adjusted ORs for CRS per 1-level change were 1.57 (95% CI, 1.27-1.96) and 1.39 (1.07-1.81), respectively. No interactions between SHS exposures and potential confounders were statistically significant.

Table 4. Unadjusted and Adjusted Conditional Logistic Regression ORs for Secondhand Smoke From All Sources Combined[a]

Variable	No. (%) Control Patients (n=306)	No. (%) Case Patients (n=306)	Unadjusted Conditional Logistic OR (95% CI)	*P* Value	Adjusted[b] Conditional Logistic OR (95% CI)	*P* Value
Exposure to SHS from any of 4 sources[c]						
No	163 (53.3)	97 (31.7)	NA	NA	NA	NA
Yes	143 (46.7)	209 (68.3)	2.35 (1.68-3.28)[d]	<.001	2.20 (1.51-3.20)[d]	<.001
Summary score: exposure from the 4 primary sources[c]						
L0 exposures	163 (53.3)	97 (31.7)	1 [Reference]		1 [Reference]	
L1 exposure	112 (36.6)	120 (39.2)	1.72 (1.20-2.47)[e]	.003	1.71 (1.15-2.56)[e]	.009
L2 exposures	28 (9.2)	76 (24.8)	4.88 (2.83-8.42)[f]	<.001	4.74 (2.49-9.02)[f]	<.001
L3 or L4 exposures[g]	3 (1.0)	13 (4.2)	10.70 (2.79-41.08)[h]	.001	9.25 (1.79-47.76)[h]	.008
1-Level change	NA	NA	2.09 (1.65-2.63)	<.001	2.03 (1.55-2.66)	<.001

Abbreviations: CI, confidence interval; L, level; NA, not applicable; OR, odds ratio; SHS, secondhand smoke.
[a] Percentages may not total 100% owing to rounding.
[b] The model was adjusted for (1) exposure to air pollution; exposure to chemicals or respiratory irritants (2) at work, (3) through hobbies, and from (4) nonwork and nonhobby sources; and (5) socioeconomic status estimated by individual educational level.
[c] This score is obtained by adding 1 for each exposure an individual had for each of home, work, and private exposures and 1 for exposure in public places if more than 10 exposures were reported per month.
[d] OR for yes vs no.
[e] OR for L1 vs L0.
[f] OR for L2 vs L0.
[g] No controls and only 2 case patients had all 4 sources of exposure; therefore, levels 3 and 4 were pooled.
[h] OR for L3/L4 vs L0.

POPULATION-ATTRIBUTABLE RISK

The SHS exposure from any of the 4 primary sources (SHS score of ≥1) was reported by 143 controls (46.7%). If the association between SHS exposure and CRS were causal, the population-attributable risk for CRS from SHS (score ≥1 vs 0) was 38.7%. When the SHS score was treated as a multilevel exposure, the population-attributable risk was 41.5%.

COMMENT

Our study found that many individuals in the Detroit population were exposed to SHS in numerous settings. In this study, population exposures to SHS are best estimated by control exposures. Control SHS exposures at home (9.1%) and work (6.9%) were relatively infrequent. Of controls, 27.8% reported exposure to SHS at private social functions, indicating that such venues are important and previously underappreciated sources of SHS exposure that might not be captured in traditional SHS surveys. Control SHS exposure in public places was high (84.3%). On the basis of an analysis of serum cotinine concentrations in nonsmoking participants in the National Health and Nutrition Examination Survey,[22] 43% of individuals (aged 4 years or older) in 2001-2002 were exposed to SHS. These statistics suggest that our study respondents may have reported SHS exposure in public places when they were exposed to SHS concentrations that would have been too low to be detectable by cotinine assay. Alternatively, public exposure to SHS in Detroit may be greater than the US national average.

Our study provides strong evidence of an association between SHS and CRS. Multivariable analyses demonstrated strong independent dose-response associations between overall SHS exposure and CRS (Table 4 and Figure). The OR estimating the association between exposure to SHS *at home* and CRS was elevated and demonstrated a trend to significance in unadjusted and adjusted analysis. *Work exposure* to SHS was significantly associated with CRS in both levels of analysis and demonstrated a dose-response relationship in unadjusted and potential confounder-adjusted analysis. *Private social exposure* to SHS as a dichotomous variable was significantly associated with CRS in both levels of analysis.

Exposure to SHS *in public places* demonstrated a significant dose-response relationship with CRS in unadjusted analysis and an elevated nonsignificant association in the potential confounder-adjusted model. However, as discussed herein, it may be that public exposure to SHS was being overreported; when exposure to SHS in public places was defined as exposure averaging more than 10 times a month, the OR was nonsignificantly elevated in the fully adjusted model.

This study has several strengths. The study case definition was based on well-established clinical criteria. Diagnosis was made within 1 department in 1 medical institution and was confirmed by computed tomography and/or endoscopy. Although SHS measurement was limited to self-reported exposures, we used an established reliable questionnaire, which quantified exposure from multiple sources and included measures of intensity. Many past studies have focused on SHS exposure at home, at work, and in public places. Our study added another important source—private social functions. Studies of the SHS-CRS association may be vulnerable to confounding. In this study, we made careful measurements and adjustments for multiple potential confounders, thus minimizing possible confounding. It is unlikely that residual confounding would account for the dose-response SHS-

CRS effects observed in this study. The HFHS population is representative of the Detroit population, and thus study sampling is approximately population based. This approach yields estimates of exposure in controls that reflect exposures in the Detroit population, and because the study SHS-CRS association is putatively biologic, we expect that our findings are generalizable to populations beyond Detroit.

Several study limitations exist. The retrospective study design is vulnerable to recall bias. Although case patients reported some exposures with a greater frequency than controls, this finding was not universal. For instance, no significant difference was found between case patients and controls in the amount of vegetables, fruit, or alcohol consumed. To attempt to minimize recall bias, study participants were masked to the hypothesis under study and SHS questions were mixed with other health-related questions. The controls had been seen at the HFHS within the last 5 years and thus may also have had conditions that might have led to more intense recall. Data regarding specific CRS etiologies were not available, so it was not possible to determine whether SHS-CRS associations differed by etiologic subtype. Biologic measurements of SHS exposure, such as serum or urine cotinine, were not available.

Only a few other studies have investigated the SHS-CRS association. Lieu and Feinstein,[10] using self-reported sinusitis and SHS exposure data from the Third National Health and Nutrition Examination Survey, 1988-1994, did not find an association between SHS and CRS. However, their cross-sectional survey design and suboptimal measures of exposure and outcome limit their findings.

Biologic mechanisms explaining an SHS-CRS association are not known with certainty, but several possibilities exist. Studies[23-26] have shown that SHS exposure increases susceptibility to respiratory infections or worsens infection in adults and children. In addition, SHS can inhibit T-cell–dependent and T-cell–independent antibody responses[27] and can impair macrophage responsiveness,[28] leading to reduced immune function. Also, SHS can impair nasal mucociliary clearance,[29,30] increase bacterial adherence, and disrupt respiratory epithelium cells.[31,32] Exposure to SHS appears to increase epithelial permeability to environmental allergens, which can enhance allergic reactions to inhaled allergens.[33,34]

Even though more evidence is needed to validate the SHS-CRS association, SHS is already known to cause many other diseases.[5,6] Thus, there is already ample reason for taking action to eliminate exposure to SHS. The US Surgeon General recommends that physicians routinely ask their patients about SHS exposure.[6] On the basis of our findings, physicians should recommend that patients who are susceptible to CRS or who have CRS avoid exposure to SHS. The dose-response relationship between SHS and CRS indicates that even modest levels of exposure carry some risk. Our findings also suggest that physicians, public health officials, and policy makers should pay greater attention to the exposure of individuals to SHS at private social functions while continuing to address exposure at home, at work, and in public places. The findings of this study need to be confirmed in other populations. It would be particularly informative to assess this association using longitudinal study designs.

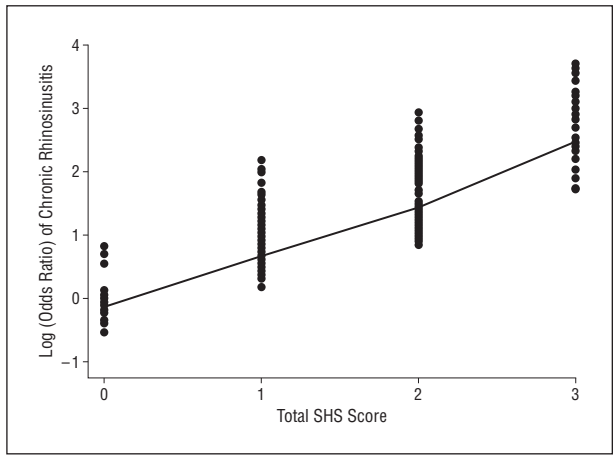

Figure. Lowess smoother describing the association between chronic rhinosinusitis and total secondhand smoke (SHS) score, adjusted for 5 potential confounders. The model was adjusted for (1) exposure to air pollution; exposure to chemicals or respiratory irritants (2) at work, (3) through hobbies, and (4) from nonwork and nonhobby sources; and (5) socioeconomic status estimated by individual educational level. Circles are individual data points; line is the Lowess line. Bandwidth is 0.8.

In conclusion, our study found that exposure to SHS is still common, and exposure through private social functions is important. Exposure to SHS is associated with CRS in a dose-response fashion and independently of environmental and occupational exposures. Assuming a causal relationship, elimination of exposure to SHS could prevent approximately 40% of CRS cases.

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