

Passive Smoking Exposure*

A Risk Factor for Chronic Bronchitis and Asthma in Adults?

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Objective: The effects of passive smoke exposure on respiratory health are still under debate. Therefore, we examined the risk of respiratory symptoms related to passive smoke exposure among German adults within the European Community Respiratory Health Survey.

Methods: The questionnaire data of the population-based sample (n = 1,890) were analyzed. Multiple logistic regression models were carried out for current asthma (asthma symptoms or medication), chronic bronchitis (cough with phlegm for ≥ 3 months per year), and wheezing as dependent variables, and self-reported exposure to passive smoke at home and at the workplace as independent variables after adjusting for city, age, gender, active smoking, and socioeconomic status as well as occupational exposure to dusts and/or gases.

Results: The relative odds for chronic bronchitis were significantly higher in subjects reporting involuntary tobacco smoke exposure in the workplace (odds ratio [OR], 1.90; 95% confidence interval [CI], 1.16 to 3.11). Likewise, the adjusted OR for asthma was slightly elevated (OR, 1.51; 95% CI, 0.99 to 2.32). The risk of chronic bronchitis (OR, 3.07; 95% CI, 1.56 to 6.06), asthma (OR, 2.06; 95% CI, 1.07 to 3.97), and wheezing (OR, 2.12; 95% CI, 1.25 to 3.58) increased significantly with a daily exposure of > 8 h.

Conclusion: The control of passive smoke exposure in the workplace might reduce the risk of respiratory symptoms independently of exposure to other airborne contaminants.

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Key words: adults; cross-sectional survey; environmental air pollutants; epidemiology; European Community Respiratory Health Survey; occupations

Abbreviations: CI = confidence interval; ETS = environmental tobacco smoke; OR = odds ratio

The prevalence of passive smoke exposure at home and at the workplace is still high throughout Europe.^{1,2} In Germany, environmental tobacco smoke (ETS) has been classified as carcinogenic,³ and passive smoke exposure in the workplace is regulated by law in recreation rooms.⁴ Because of the time spent at the workplace and in the residential

setting, these environments are considered to be the most important places of involuntary tobacco smoke exposure.⁵ Especially for those without exposure to tobacco smoke in the home environment, the workplace is the major contributor to passive smoke exposure.⁶

While data on the association between passive smoke exposure during childhood and respiratory health are consistent,^{7–11} the evidence for the development and exacerbation of asthma and chronic bronchitis by chronic exposure to ETS in adults is limited and inconclusive.^{2,12–18} Although epidemiologic studies^{2,5,15,16,19,20} on the association between exposure to passive smoke and asthma support the hypothesis that ETS exposure may increase the risk of asthma in adults, the results of controlled exposure studies^{12,21,22} among asthmatic patients were inconsistent.

Most of the epidemiologic studies⁵ did not control for the potential confounding factor of exposure to

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other airborne contaminants in the workplace. Other studies¹⁹ failed to take into account passive smoke exposure in the home environment. Therefore, the aim of this secondary analysis of the data set of the European Community Respiratory Health Survey in Germany was to assess whether exposure to passive smoke is associated with an increased risk of chronic bronchitis and asthma in young adults after adjusting for workplace exposure and other potential confounding variables. Additionally, we examined the dose-response relationship between the daily duration of passive smoking exposure and the risk of chronic bronchitis and asthma.

MATERIALS AND METHODS

Subjects

Within the European Community Respiratory Health Survey, a population-based, cross-sectional survey including 55 centers in 23 countries,²³ a representative sample of 4,500 subjects in Hamburg, Germany, and 4,990 subjects in Erfurt, Germany, who were 20 to 44 years of age was obtained from the offices of the population census. In stage I of the study, a one-page screening questionnaire was mailed to all subjects (response rate, 76.8%). All eligible subjects in Hamburg ($n = 4,090$) and a random sample of the stage I responders in Erfurt ($n = 1,338$) were invited to participate in the second stage of the study, including a 71-item interview-based questionnaire, spirometry, methacholine challenge, and skin testing. Only the data of the long questionnaire were used in this analysis (response rate, 42.1%).

Long Questionnaire

The detailed questionnaire²³ contained questions on smoking exposure, respiratory symptoms, and occupational exposure to gases, fumes, and/or dusts. Based on the responses to the questionnaire, *asthma* was defined as a positive answer to at least one of the following questions²⁴:

1. Are you currently taking any medicine (including inhalers, aerosols, or tablets) for asthma?
2. Have you been awakened by an attack of shortness of breath at any time in the last 12 months?
3. Have you had an attack of asthma in the last 12 months?

Chronic bronchitis was defined as the existence of cough and phlegm for at least 3 months per year. *Wheezing* was defined as wheezing during the last 12 months without having a cold.

The following question on second-hand smoking exposure at home and at the workplace was asked of the participants²³: "Have you been regularly exposed to tobacco smoke in the last 12 months [*regularly* means on most days or nights]?"

If the answer to that question was "yes," the following questions were asked:

1. Not counting yourself, how many people in your household smoke regularly?
2. Do people smoke regularly in the room where you work?
3. How many hours per day are you exposed to other people's tobacco smoke?

Respondents with at least a high school degree were classified as having *higher educational level*.

The occupational exposure to biological dusts, mineral dusts, gases, and fumes was based on a job-exposure matrix.²⁴ The most recent job reported by the participants was coded according to the European Community socioeconomic status groups classification.²⁵ Based on the job-exposure matrix, each of the 350 occupational types of this coding scheme then was classified as *not exposed* or *exposed* to each of the three groups of airborne pollutants.

Statistical Analysis

Logistic regression models were carried out to assess the association between passive smoke exposure and respiratory symptoms. These models were adjusted for city, age, gender, active smoking, socioeconomic status, and occupational exposure to gases, fumes, and/or dusts. Additionally, a potential effect modification by active smoking, gender, and occupational exposure to gases, fumes, and/or dusts was assessed using interaction terms.

In order to assess a dose-response relationship, subjects reporting any exposure to passive smoke were classified into four exposure categories based on the quartiles of self-reported daily duration of passive smoke exposure (1, > 0 to ≤ 2 h per day; 2, > 2 h to ≤ 4 h per day; 3, > 4 h to ≤ 8 h per day; 4, > 8 h per day). Unexposed subjects were used as a reference category. Odds ratios (ORs) with 95% confidence interval (CIs) were computed, adjusting for city, age, gender, socioeconomic status, active smoking, and occupational exposure to gases, fumes, and/or dusts.

RESULTS

The study population in its relationship to passive smoking exposure is described in Table 1. Most participants reporting involuntary tobacco smoke exposure were exposed at home and at the workplace (62.9%), with a mean (\pm SD) duration of 5 ± 4 h per day. Subjects reporting passive smoke exposure were less likely to have a higher educational level and to live in Erfurt. Furthermore, they were more likely to be exposed to gases and/or mine dusts at the workplace. Additionally, subjects with current passive smoking exposure reported a higher prevalence of ETS exposure during childhood. The crude comparison between passive smoking exposure and prevalence of asthma or chronic bronchitis did not show any significant difference. However, the 12-month prevalence of wheezing was significantly higher among those who reported passive smoking exposure (12.8% vs 7.5%, respectively).

The results of the multiple logistic regression models are given in Table 2. After adjusting for city, age, gender, socioeconomic status, and smoking habits, the OR for asthma was increased by passive smoking exposure at the workplace (OR, 1.51; 95% CI, 0.99 to 2.32). Additionally, a slight increase in the relative odds for asthma was seen with occupational exposure to mineral dusts. The association between passive smoke exposure at the workplace and chronic bronchitis was even more pronounced (OR, 1.90;

Table 1—Descriptive Data in Relation to Passive Smoke Exposure at Home and/or in the Workplace*

Variables	Passive Smoke Exposure	
	Yes (n = 909)	No (n = 972)
Hamburg location	576 (63.4)	577 (59.4)
Erfurt location	333 (36.6)	395 (40.6)
Age, yr	32.0 ± 6.9	33.1 ± 6.8
Female gender	458 (50.4)	463 (47.6)
Higher educational level	359 (39.8)	498 (51.4)
Occupational exposure		
Gases/fumes	270 (29.9)	256 (26.6)
Biological dust	124 (13.7)	111 (11.6)
Mineral dust	217 (24.1)	195 (20.3)
Current smokers	515 (56.7)	313 (32.2)
Ex-smokers	168 (18.5)	251 (25.8)
ETS exposure during childhood		
Father smoked	596 (72.1)	556 (61.1)
Mother smoked	272 (30.7)	230 (24.2)
Current ETS exposure		
Only at home	286 (20.4)	
Only at the workplace	234 (16.7)	
At home and workplace	883 (62.9)	
Daily duration of passive smoking exposure, h†	5.0 ± 3.7	0 ± 0
Respiratory symptoms		
Asthma	51 (5.6)	52 (5.3)
Chronic bronchitis	42 (4.6)	31 (3.2)
Wheezing	116 (12.8)	73 (7.5)

*Values given as No. (%) or mean ± SD.

†Including only those subjects with passive smoking exposure.

95% CI, 1.16 to 3.11). Moreover, the risk of reporting symptoms of chronic bronchitis was doubled if the subject reported occupational exposure to biological dusts. The risk of wheezing was significantly associated with neither passive smoking exposure at the workplace (OR, 1.26; 95% CI, 0.91 to 1.75) nor at home (OR, 1.13; 95% CI, 0.81 to 1.60). Including passive smoking exposure during childhood did not change the risk estimates (data not shown). Including interaction terms in the logistic regression models did not give any indication for effect modification by active smoking, gender, or occupational exposure.

Taking the daily duration of passive smoke exposure into account, dose-response relationships could be established (Fig 1). The OR for asthma was significantly increased for subjects with > 8 h of self-reported exposure to passive smoke per day (OR, 2.06; 95% CI, 1.07 to 3.97). Likewise, the relative odds for chronic bronchitis (OR, 3.07; 95% CI, 1.56 to 6.06) and wheezing (OR, 2.12; 95% CI, 1.25 to 3.58) was significantly increased for the highest exposure category.

DISCUSSION

In this study, involuntary tobacco smoke exposure, especially in the workplace, was associated with the prevalence of respiratory symptoms in young adults even after adjustment for occupational exposure. The risk estimates increased significantly with the increasing duration of daily exposure to second-hand smoke.

Our study has several strengths. Since it is a population-based survey, it reflects the prevalence of respiratory symptoms as well as the exposure to ETS exposure in the general population. The information on exposure, outcome, and potential confounding variables was assessed in detail with a validated questionnaire instrument.²³ Therefore, the analyses could be adequately controlled for potential confounding variables. Additionally, the dose-response relationship seen between the daily duration of exposure and respiratory health supports the findings of the survey. Moreover, we had the opportunity to take into account occupational exposure to other substances that potentially are associated with respiratory morbidity.²⁴

However, some limitations to the study have to be considered. Since the survey was performed cross-sectionally, the temporality of the results cannot be estimated. Symptoms might result in a change of exposure patterns. This would result in a bias toward the null. Our finding that subjects without any exposure to passive smoke had a slightly higher risk

Table 2—Passive Smoking Exposure, Occupational Exposure, and Respiratory Symptoms*

Exposure	Asthma* (n = 1,843)	Chronic Bronchitis* (n = 1,844)	Wheezing* (n = 1,841)
Passive smoke exposure			
At home	0.95 (0.59–1.52)	0.82 (0.48–1.42)	1.13 (0.81–1.60)
At the workplace	1.51 (0.99–2.32)	1.90 (1.16–3.11)	1.26 (0.91–1.75)
Occupational exposure			
Gases, fumes	0.66 (0.36–1.20)	0.66 (0.33–1.30)	1.36 (0.90–2.07)
Biological dusts	1.05 (0.53–2.09)	2.04 (1.01–4.15)	0.90 (0.54–1.51)
Mineral dusts	1.60 (0.83–3.08)	0.73 (0.34–1.58)	0.95 (0.59–1.53)

*Values given as OR (95% CI). Values were adjusted for city, age, sex, level of education, and active smoking, and were mutually adjusted for environmental tobacco smoke and occupational exposure.

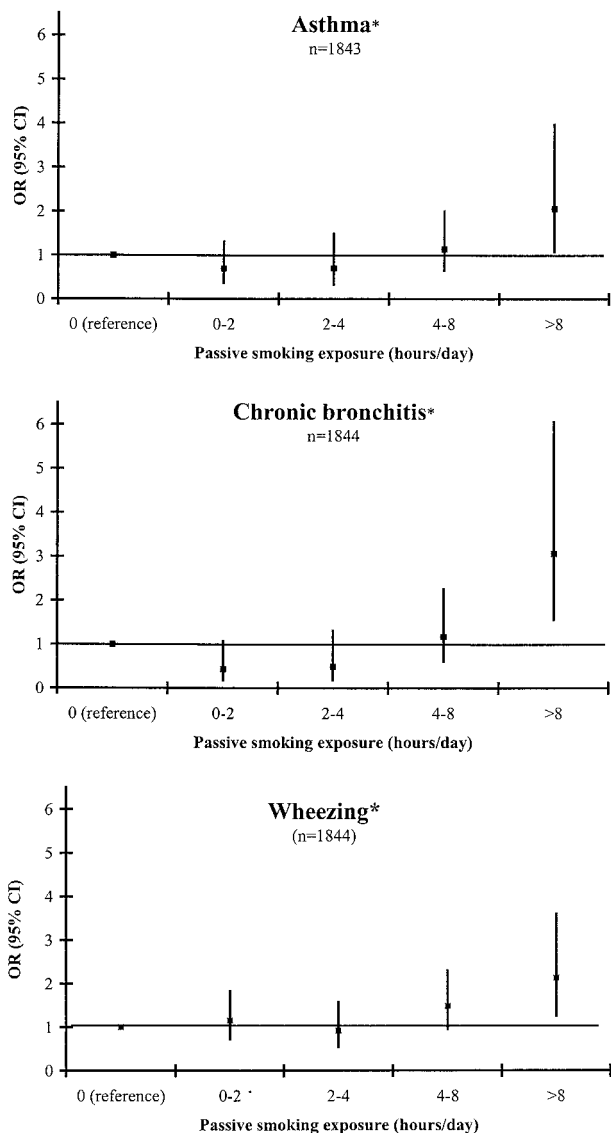


FIGURE 1. Dose-response relationship between the daily duration of passive smoking exposure and respiratory symptoms. *Values were adjusted for city, age, sex, level of education, active smoking, and occupational exposure.

of symptoms compared to subjects with moderate exposure supports this hypothesis. In contrast, subjects with respiratory symptoms may be more likely to report exposure. Since no objective measurements of passive smoking exposure were performed in our study, the accuracy of the self-reported exposure is difficult to estimate. However, previous studies²⁶ have shown a high validity of self-reported exposure to ETS. Among smokers, second-hand exposure to tobacco smoke in the workplace might be a proxy for their amount of active smoking since these smokers themselves have the opportunity to smoke at the workplace. Overall, misclassification of exposure cannot be excluded in the present study.

The relatively low response rate might have resulted in a higher participation rate of symptomatic subjects.^{27,28} Comparing the age and gender distribution of the participants with the distribution in the target population, no differences with the target population have been observed.²⁹ However, we could not perform a nonresponder analysis based on socioeconomic status or passive smoke exposure since we did not have any information on these parameters for nonresponders. Considering that it is unlikely that the response rates differed by exposure to ETS, it is not likely that our risk estimates were essentially overestimated due to nonresponder bias.

Some residual confounding might have occurred since subjects with passive smoke exposure may have different characteristics than subjects without such an exposure.³⁰ Additionally, atopy might be considered a confounder for the association between passive smoking exposure and asthma.³¹ However, in restricting the analysis to subjects who underwent a skin-prick tests to common aeroallergens ($n = 1,612$), the results of the logistic regression models for asthma did not change (data not shown).

We did not find any indications for effect modification by active smoking, gender, or occupational exposure. However, the number of symptomatic subjects in each stratum was low. For instance, only 23 nonsmoking subjects reported symptoms of chronic bronchitis. Additionally, due to the limited number of symptomatic subjects in each stratum no stratified analyses could be performed.

Our finding that exposure to tobacco smoke in the workplace is a stronger predictor for respiratory morbidity than tobacco smoke exposure in the home environment is consistent with those of previous studies.^{2,12} The exposure levels in the workplace have been estimated to be higher than in the home environment,³² and the time spent in the work environment might be longer.² As a result, occupational exposure to ETS might be considered an occupational health concern with respect to respiratory morbidity. Our findings indicate that there is a need for regulating tobacco smoke exposure, especially in the workplace. This might be true even for workplaces with occupational exposure to gases and/or dusts.

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