



Socioeconomic Status and Asthma Prevalence in Young Adults

The European Community Respiratory Health Survey

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The authors assessed the association between asthma prevalence and socioeconomic status at both the individual and center levels simultaneously by using data from 32 centers in 15 countries. Included were 10,971 subjects aged 20–44 years selected from the general population and interviewed in 1991–1992. Socioeconomic status at both the individual and aggregated levels was measured on the basis of occupation and educational level. Associations were assessed by using multilevel models adjusted for age, sex, body mass index, parental asthma, childhood respiratory infections, presence of immunoglobulin E to common allergens, rhinitis, smoking, and occupational exposure to irritants. Asthma prevalence was higher in lower socioeconomic groups, whether defined by educational level (odds ratio for finishing full-time studies—<16 vs. >19 years = 1.28, 95% confidence interval: 1.00, 1.64) or social class (odds ratio for semiskilled and unskilled manual workers vs. professional/managerial = 1.51, 95% confidence interval: 1.20, 1.90), regardless of atopic status. The relation was consistent between centers. Irrespective of individual socioeconomic status, subjects living in areas in which educational levels were lower had a higher risk of asthma ($p < 0.05$). This center-level association partially explained geographic differences in asthma prevalence, but considerable heterogeneity still remained. The authors concluded that community influences of living in a low-educational area are associated with asthma, independently of subjects' own educational level and social class.

adult; asthma; education; prevalence; social class

Abbreviations: ECRHS, European Community Respiratory Health Survey; SES, socioeconomic status.

The relation between socioeconomic status (SES) and asthma in adults is not well understood. Studies have shown increased asthma hospital admissions for those who are materially deprived (1, 2) and increased asthma severity in low social class groups (3). However, the association between socioeconomic factors and asthma prevalence is less clear.

Existing studies are heterogeneous regarding the definition of asthma and the socioeconomic indicators used. A negative association between asthma prevalence and SES was found in most studies using SES measures based on occupation, income, or education (4–10), but not in all (11, 12). Further-

more, associations vary depending on whether asthma is defined as “atopic” or “nonatopic” (12, 13). In both the United States (12) and England (4), low educational level or social class has been associated with nonatopic asthma, while high educational level has been associated with atopic asthma.

Asthma may provide an excellent paradigm for understanding the role of contextual factors in disease (14). Distribution of asthma shows strong geographic and temporal variations that remain unexplained by known risk factors, which led to reconsideration of the interplay with social determinants (14). Community-level variables linked to

asthma include some of the following: environmental exposures such as air pollution; physical and psychological demands of living in a relatively deprived environment that may potentiate a person's susceptibility to environmental exposures (14); characteristics of the community, such as degree of social support or exposure to poverty, that may influence chronic life stress, which has been suggested to affect asthma (14); and community beliefs and practices about health that may affect access to health care and treatment practices (14). A relation of asthma to a community characteristic may reflect a direct association or may be the combined effect of a set of unmeasured individual characteristics for which that area-level variable is a proxy. In addition, an area-level variable may modulate individual-level relations. Certain studies examined the relation between prevalence of asthma and poverty by area of residence and found an increase in the prevalence of asthma among adults living in the most deprived areas (7, 15, 16). To elucidate whether group-level exposures are related to outcomes beyond the effect of individual-level exposures, it is necessary to conduct multilevel studies. To our knowledge, such studies have never been conducted to explain adult asthma in an international setting.

We assessed the association between asthma prevalence and SES simultaneously at the individual and area levels by using data from the European Community Respiratory Health Survey (ECRHS). Areas were defined by preexisting administrative boundaries with a population of at least 150,000 inhabitants (17).

MATERIALS AND METHODS

Study population and questionnaire

The ECRHS multicenter, cross-sectional study was carried out in 1991–1992 among subjects aged 20–44 years randomly selected from the general population. The methodology used for the ECRHS has been described elsewhere (17). The institutional review boards of the participating centers approved the study protocol, and participants gave informed written consent. In this analysis, data for a random general population sample from 32 study centers were included. Centers were located in Europe, Australia, New Zealand, and the United States. Differences in the response rates between the study centers have been noted previously (18). Information on asthma, SES, and selected confounding variables was available for 10,971 persons (58 percent of those eligible, from 20 percent in France to 89 percent in Sweden). The most frequent reason for incomplete information was refusal to provide a blood sample for immunoglobulin E testing. Sensitivity analysis of the effect of nonresponse in ECRHS showed a minimal influence (18).

Information on respiratory symptoms, self-reported asthma and allergic disorders, environment, and lifestyle was collected by using an interviewer-led questionnaire (17). Current asthma was defined as the presence of at least one of the following factors in the last 12 months: 1) being awakened by an attack of shortness of breath, 2) having an asthma attack, and 3) currently using medication for asthma (17). Asthma was stratified according to the presence of atopy (4,

12, 19), defined as specific sensitization (specific immunoglobulin E > 0.35 kU/liter) to at least one of the common allergens *Dermatophagoides pteronyssinus*, Timothy grass, cat, or *Cladosporium herbarum*.

Information on individual SES was derived from self-reported occupation and education. Current or last-held reported occupation was categorized according to the European Community SES groups classification, and social class was coded by using the British Registrar General's Scale (20). A group of "not classifiable" included students and housewives. Level of education was categorized by age at completion of full-time studies, an approach found to be reasonably satisfactory for comparing educational achievement between populations (21), and was defined as low (age at completion <16 years), medium (age 16–19 years), and high (age ≥20 years). Selecting a lower cutoff for the low-education group resulted in no observations in this category for some centers. The other two categories were defined to include a similar number of observations. The SES of the residential area was determined by calculating the proportion of all subjects from the corresponding study center classified as of high and low social class and as having high and low levels of education.

All risk factors previously associated with asthma in any of the ECRHS studies were controlled for in our models as possible confounders (22, 23). Individual-level variables were age, sex, body mass index, family history of asthma (defined as at least one parent having had asthma), smoking, passive smoking, number of siblings, respiratory infections before age 5 years, rhinitis, mold or mildew in the home in the last 12 months, and occupational exposure to biologic or mineral dusts, gases, or fumes—information obtained by using a job-exposure matrix described elsewhere (24).

Statistical methods

The associations between asthma and socioeconomic variables were estimated by using multilevel (or hierarchical) models. These models are indicated when there is a hierarchical structure in levels of data, with a single dependent variable measured at the lowest level and a set of explanatory variables on each of the levels. The advantage of these models lies in their capacity to reveal and define variations at each level of the hierarchy after controlling for relevant explanatory variables (25–27). Since the response regarding the presence or absence of asthma is binary, we fitted multilevel logistic models (25).

We followed the model-building procedure proposed by Hox (28). We first entered individual-level explanatory variables as fixed effects, and only those with a *p* value of <0.10 in the adjusted model were retained. Next, we assessed whether any of the coefficients of any of the explanatory variables had a significant variance component between centers (heterogeneity of the effect). After that, we added center-level explanatory variables to determine whether these variables explained between-center variation in the dependent variable. Finally, we tested cross-level interactions in the variables for which there was heterogeneity in their coefficients.

TABLE 1. Description of outcome and socioeconomic variables used to assess socioeconomic status and asthma prevalence in young adults, European Community Respiratory Health Survey, 1991–1992

Individual variables	No.	%	Range (%) by center
Outcome variables			
Asthma	917	8.4	2.8–15.7
Atopic	512	4.7	1.1–10.7
Nonatopic	405	3.7	1.3–8.8
No asthma	10,054	91.6	84.3–97.2
Atopic	3,128	28.5	13.9–37.2
Nonatopic	6,926	63.1	49.0–77.3
Socioeconomic variables			
Social class*			
I–II	4,035	36.8	18.1–60.1
III nonmanual	2,308	21.0	9.1–33.3
III manual	2,278	20.8	10.4–33.3
IV–V	1,703	15.5	3.7–38.9
Unclassifiable	647	5.9	0–16.8
Educational level (age at which studies were finished)			
High (≥ 20 years)	4,580	41.7	12.1–65.7
Middle (16–19 years)	4,901	44.7	32.0–70.7
Low (< 16 years)	1,490	13.6	0.4–43.6
High occupational exposure to biologic or mineral dust, gas, or fumes			
	1,446	13.2	5.3–22.9
Mold or mildew in the home in the last year			
	2,409	22.0	4.4–57.1

* I, professional; II, managerial; III nonmanual, clerical; III manual, skilled manual; IV, semiskilled manual; V, unskilled manual.

The resulting odds ratios for the individual-level variables are conditional on the random effects. The odds ratio for cluster-level variables was computed for a change from the 25th percentile to the 75th percentile of the observed distribution of that variable. In multilevel logistic models, the variance of the random effects is difficult to interpret as a heterogeneity measure, so we also reported a more interpretable measure, the median odds ratio (29). This measure can be considered the median of the odds ratios that we would obtain by choosing two subjects with the same covariates but from different clusters (centers in our case), computing the odds ratio between them (with data for the person from the area with a higher propensity in the numerator), and repeating this procedure many times. Because we always put data for the person at higher risk in the numerator, the odds ratios, and consequently the median odds ratio, will always be greater (or equal) than 1. A value of the median odds ratio far greater than 1 indicated substantial heterogeneity between clusters, and this value is directly comparable to fixed-effects odds ratios. Thus, a median odds ratio of 2 compared with an odds ratio of 1.5 for a cluster-level variable indicates that the remaining between-cluster heterogeneity

is of a higher magnitude than the effect of the cluster-level variable (29).

Models were fitted by using MLwiN 1.1 software (30). Models were replicated with MIXOR version 2 software (31), which uses numerical integration to produce a valid estimation of the likelihood and allows use of likelihood-ratio tests to compare nested models. Estimations did not vary to a great extent between software packages.

RESULTS

The overall prevalence of asthma was 8.4 percent, ranging from 2.8 percent to 15.7 percent across the centers (table 1). The overall prevalence of atopic asthma was 4.7 percent. Most study participants belonged to higher social classes and were categorized as having achieved a medium or high educational level, but there was considerable variation by center regarding these variables. The percentage of high social class (professional and managerial occupations) ranged from 18 percent (Galdakao, Spain) to 60 percent (Melbourne, Australia), and the percentage of high education ranged from 12 percent (Hawkes-Bay, New Zealand) to 66 percent (Bergen, Norway). The distribution of variables

TABLE 2. Distribution (%) of individual variables by asthmatic/atopic status, European Community Respiratory Health Survey, 1991–1992

Variables	No asthma (n = 10,054)	Asthma (n = 917)	Asthma and atopy (n = 512)	Asthma and no atopy (n = 405)
Age (years)				
<30	31.4	31.3	34.6	27.1
30–39	43.1	41.3	42.2	40.2
≥40	25.6	27.4	23.2	32.8
Sex: female	49.3	55.7	52.0	61.5
Body mass index (kg/m ²) (mean)	24.25	24.99	24.80	25.24
Family history of asthma	10.4	22.4	24.4	19.8
Smoking				
Nonsmoker	42.2	41.6	44.0	38.5
Former smoker	21.3	23.0	24.6	21.0
Smoker: <20 cigarettes/day	22.4	21.3	19.5	23.5
Smoker: ≥20 cigarettes/day	14.1	14.2	11.9	17.0
Passive smoking	56.9	57.1	55.1	59.8
No. of siblings				
0	38.7	38.3	41.6	34.1
1	29.4	29.4	31.1	27.4
≥2	31.9	32.3	27.3	38.5
Respiratory infections before age 5 years	9.5	16.0	17.4	14.3
Sensitized to <i>Dermatophagoides pteronyssinus</i>	17.4	36.3	65.0	0
Sensitized to cat dander	7.2	22.9	41.0	0
Sensitized to grass	17.2	34.8	62.3	0
Sensitized to <i>Cladosporium herbarum</i>	2.7	6.7	11.9	0
Rhinitis	22.1	52.1	69.5	30.1
Mold or mildew in the home in the last year	21.4	28.6	29.3	27.7
Social class*				
I–II	37.2	32.6	35.4	29.1
III nonmanual	20.9	22.7	22.3	23.2
III manual	20.9	19.2	18.2	20.5
IV–V	15.2	18.9	18.2	19.8
Unclassifiable	5.8	6.7	6.1	7.4
Educational level				
High	42.2	36.3	40.4	31.1
Medium	44.5	46.6	48.4	44.2
Low	13.3	17.1	11.1	24.7
Occupational exposure to biologic or mineral dust, gas, or fumes	13.0	15.3	12.9	18.3

* I, professional; II, managerial; III nonmanual, clerical; III manual, skilled manual; IV, semiskilled manual; V, unskilled manual.

at the individual level according to asthmatic and atopic status is shown in table 2. Among asthmatics, there was a higher proportion of women; body mass index was higher; and there were higher proportions of subjects with a family history of asthma, respiratory infections before the age of 5 years, allergic sensitization, rhinitis, and mold in the house and of subjects of low social class and low educational level.

Compared with asthmatics without atopy, asthmatics with atopy had a higher educational level and a lower exposure to occupational pollutants.

Subjects in high social classes included a higher proportion of the older age group, included fewer active and passive smokers, had fewer siblings, had a greater likelihood of sensitization to *C. herbarum*, and had a higher prevalence

TABLE 3. Distribution (%) of individual variables by social class and education groups, European Community Respiratory Health Survey, 1991–1992

Variables	Social class*					Educational level		
	I–II	III nonmanual	III manual	IV–V	Unclassifiable	High	Medium	Low
Age (years)								
<30	24.6	33.3	33.5	36.6	44.8	32.4	34.9	16.4
30–39	45.5	43.5	42.1	40.2	34.6	43.9	42.5	41.2
≥40	29.9	23.2	24.4	23.3	20.6	23.7	22.6	42.4
Sex: female	49.5	66.8	25.9	52.4	69.9	48.2	50.5	52.9
Body mass index (kg/m ²) (mean)	24.13	24.02	25.00	24.45	23.86	23.78	24.43	25.58
Family history of asthma	10.9	11.2	11.1	12.9	12.8	10.4	11.4	14.6
Smoking								
Nonsmoker	46.6	44.5	34.6	37.0	46.8	48.2	39.5	32.4
Former smoker	23.6	21.2	20.6	19.7	16.4	21.7	21.5	20.5
Smoker: <20 cigarettes/day	19.2	22.5	24.3	25.7	24.4	20.1	24.3	22.2
Smoker: ≥20 cigarettes/day	10.6	11.8	20.5	17.6	12.4	10.0	14.7	24.9
Passive smoking	47.4	59.2	66.1	66.1	51.2	48.8	60.6	69.4
No. of siblings								
0	41.5	38.9	35.2	35.1	42.0	42.7	38.1	28.4
1	30.4	29.8	29.8	27.5	26.1	31.2	28.8	26.2
≥2	28.1	31.3	35.1	37.5	31.8	26.2	33.1	45.4
Respiratory infections before age 5 years	10.5	10.2	9.1	9.8	11.0	10.9	9.6	8.9
Sensitized to <i>Dermatophagoides pteronyssinus</i>	19.8	17.7	20.4	17.7	15.8	18.4	20.0	17.0
Sensitized to cat dander	9.2	8.9	7.8	7.5	7.4	9.8	8.6	4.3
Sensitized to grass	19.8	18.3	17.7	17.6	18.7	20.3	19.1	12.0
Sensitized to <i>Cladosporium herbarum</i>	3.4	2.5	3.3	2.1	3.4	3.3	3.1	1.8
Rhinitis	27.3	25.0	21.3	21.2	26.7	26.8	23.8	20.6
Mold or mildew in the home in the last year	23.6	22.1	19.5	21.0	22.1	21.5	22.6	21.2
Occupational exposure to biologic or mineral dust, gas, or fumes	1.2	1.9	35.2	32.6	0	6.4	16.0	24.9
Educational level								
High	64.6	34.7	23.8	19.8	45.0			
Medium	30.6	54.9	56.7	53.1	31.5			
Low	4.8	10.4	19.5	27.1	23.5			

* I, professional; II, managerial; III nonmanual, clerical; III manual, skilled manual; IV, semiskilled manual; V, unskilled manual.

of rhinitis and of living in homes with mold or mildew in the last year (table 3). There was a higher percentage of women than men in the skilled nonmanual or not-classified (a category including housewives) occupational categories, but women were underrepresented in the skilled manual occupations. Higher social class groups had a lower body mass index. In addition, there were differences between social classes regarding sensitization to *D. pteronyssinus*, but no trend by social class was found. When education was used as the socioeconomic variable, all of these associations were similar, except for 1) family history of asthma, which was lower for those with a higher level of education; 2) sensitization to cat, which was higher in the higher education group;

and 3) mold in the home, which did not vary by educational level. We found a significant correlation between the variables social class, educational level, and high occupational exposure to pollutants (table 3). Spearman's correlation coefficients were 0.40 for the relation between social class and education (range by country, 0.33–0.52), 0.40 for social class and occupational exposures (range, 0.23–0.47), and 0.20 for education and occupational exposures (range, 0.07–0.34).

Results from multilevel models are presented in table 4. To assess heterogeneity by center in the absence of other explanatory variables, model 1 included only age and sex. Model 2 included the individual-level variables retained.

TABLE 4. Association (adjusted* odds ratio and 95% confidence interval) of asthma with individual- and center-level characteristics using multilevel logistic regression, European Community Respiratory Health Survey, 1991–1992

	Model 1		Model 2		Model 3	
	OR†	95% CI†	OR	95% CI	OR	95% CI
<i>Fixed effects</i>						
Individual level						
Age (years) (reference = <30)						
30–39	0.93	0.79, 1.10	0.96	0.80, 1.14	0.96	0.80, 1.15
≥40	1.05	0.87, 1.25	1.11	0.90, 1.36	1.12	0.91, 1.37
Sex: female	1.27	1.11, 1.46	1.29	1.10, 1.51	1.29	1.10, 1.50
Body mass index (kg/m ²)			1.04	1.02, 1.06	1.04	1.02, 1.06
Family history of asthma			2.00	1.66, 2.41	2.01	1.67, 2.42
Smoking (reference = nonsmoker)						
Former smoker			1.17	0.97, 1.42	1.17	0.97, 1.43
Smoker: <20 cigarettes/day			1.17	0.96, 1.43	1.17	0.96, 1.42
Smoker: ≥20 cigarettes/day			1.29	1.02, 1.62	1.29	1.02, 1.63
Respiratory infections before age 5 years			1.81	1.47, 2.23	1.80	1.46, 2.22
Sensitized to <i>Dermatophagoides pteronyssinus</i>			1.66	1.40, 1.98	1.64	1.38, 1.96
Sensitized to cat dander			2.25	1.83, 2.77	2.27	1.85, 2.79
Sensitized to <i>Cladosporium herbarum</i>			1.62	1.16, 2.25	1.61	1.15, 2.25
Rhinitis			2.87	2.45, 3.35	2.89	2.47, 3.38
Social class‡ (reference = I–II)						
III nonmanual			1.28	1.04, 1.57	1.28	1.04, 1.58
III manual			1.18	0.94, 1.48	1.18	0.94, 1.48
IV–V			1.51	1.20, 1.90	1.51	1.20, 1.91
Unclassifiable			1.18	0.85, 1.65	1.17	0.84, 1.63
Educational level (reference = high)						
Medium			1.04	0.87, 1.25	1.01	0.84, 1.20
Low			1.28	1.00, 1.64	1.21	0.94, 1.55
Center level						
Percentage of low and medium education§					1.39	1.11, 1.73
<i>Random effects</i>						
σ_u^2 (center)¶		0.174		0.118		0.077
Median odds ratio		1.49		1.39		1.30

* Variables included because of a *p* value of <0.10 for those shown in table 2. Variables not retained were passive smoking, number of siblings, sensitization to grass, mold or mildew in the home in the last year, and occupational exposure to biologic or mineral dust, gas, or fumes.

† OR, odds ratio; CI, confidence interval.

‡ I, professional; II, managerial; III nonmanual, clerical; III manual, skilled manual; IV, semiskilled manual; V, unskilled manual.

§ Odds ratio for a difference of 28% (interquartile range) in the percentage of low and medium educational levels associated with the centers.

¶ Variance of the center-level random intercept.

None of the odds ratios demonstrated significant heterogeneity between centers. Subjects of lower social classes and having less education had a higher prevalence of asthma even after we adjusted for all variables in model 2. High occupational exposure to pollutants could have been entered

in the model instead of social class or education with an equivalent goodness of fit, but only two of these three variables could remain in the model simultaneously because of the mutual correlation. When the model contained only one of the following variables—low social class, low educational

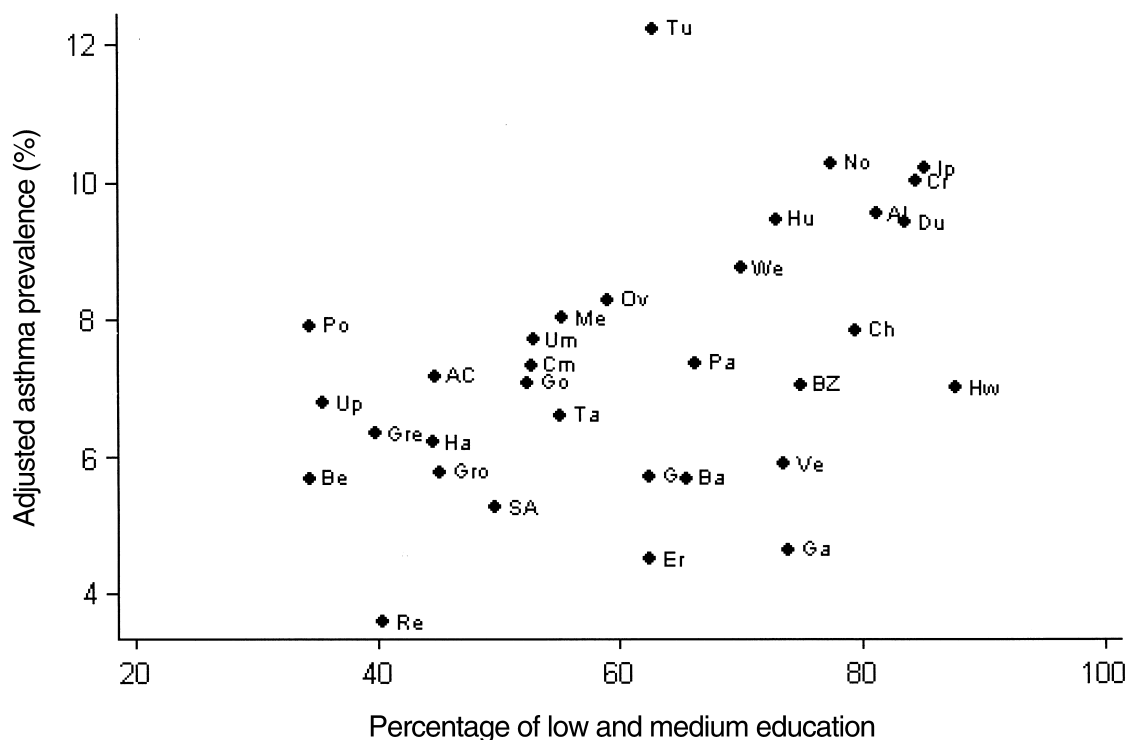


FIGURE 1. Plot of adjusted asthma prevalences for each European Community Respiratory Health Survey center (obtained by using empirical Bayes' estimates from model 2 in table 3) against the percentage of low or medium educational levels in the centers, 1991–1992. AC: Antwerp City, Belgium; Al: Albacete, Spain; Ba: Barcelona, Spain; Be: Bergen, Norway; BZ: Bergen op Zoom, the Netherlands; Ch: Christchurch, New Zealand; Cm: Cambridge, United Kingdom; Cr: Cardiff, United Kingdom; Du: Dublin, Ireland; Er: Erfurt, Germany; Ga: Galdakao, Spain; G: Geleen, the Netherlands; Go: Göteborg, Sweden; Gre: Grenoble, France; Gro: Groningen, the Netherlands; Ha: Hamburg, Germany; Hu: Huelva, Spain; Hw: Hawkes-Bay, New Zealand; Ip: Ipswich, United Kingdom; Me: Melbourne, Australia; No: Norwich, United Kingdom; Ov: Oviedo, Spain; Pa: Pavia, Italy; Po: Portland, Oregon; Re: Reykjavik, Iceland; SA: South Antwerp, Belgium; Ta: Tartu, Estonia; Tu: Turin, Italy; Um: Umeå, Sweden; Up: Uppsala, Sweden; Ve: Verona, Italy; We: Wellington, New Zealand.

level, and high occupational exposure—the odds ratios were 1.58 (95 percent confidence interval: 1.27, 1.95), 1.45 (95 percent confidence interval: 1.15, 1.83), and 1.35 (95 percent confidence interval: 1.10, 1.65), respectively.

Regarding heterogeneity, the median odds ratio for model 2 was 1.39, 7 percent lower than that for model 1 (1.49) (table 4) but indicating that a considerable heterogeneity between centers still remained; of two subjects with the same individual characteristics, one will have, in median, a 39 percent higher probability of asthma only because of area of residence. This heterogeneity could alternatively be assessed by looking at adjusted asthma prevalences for each center, obtained from model 2, which were plotted against the percentage of low and medium educational levels associated with each center shown in figure 1. This figure shows a positive relation between these two variables. Education, but not social class, was significant at the center level (model 3), demonstrating that living in a center in which more people were less educated (low and medium levels of education) was an independent risk factor for asthma. In particular, given two subjects with the same individual covariates, the one from a center with 28 percent more low and medium educational levels (the interquartile range of the distribution in the sample) would have a 39

percent higher risk of asthma. Education was more important at the center than at the individual level, indicated by the lack of significance of the individual-level education variable in this model. After including education at the center level, we found that the median odds ratio was reduced by an additional 7 percent (1.39 vs. 1.30), but considerable between-area heterogeneity still remained unexplained.

Separate multilevel models were fitted for atopic and nonatopic subjects (table 5). Because of collinearity between social class and the variables education and occupational exposure, they were tested in separate models. Low social class was positively associated with asthma in both atopic and nonatopic subjects at the individual level, as well as low educational level at the center level. However, low educational level, high occupational exposures, and household mold during the last year were positively related to asthma in nonatopics only. None of the odds ratios associated with individual-level variables showed significant heterogeneity between centers.

DISCUSSION

In this study, we showed that the prevalence of asthma in young adults was higher in lower socioeconomic groups,

TABLE 5. Association (adjusted* odds ratio and 95% confidence interval) between asthma and socioeconomic conditions among subjects with atopy and subjects without atopy, European Community Respiratory Health Survey, 1991–1992

	Asthma in atopics (n = 3,640)		Asthma in nonatopics (n = 7,331)	
	OR†	95% CI†	OR	95% CI
<i>Model 1 (socioeconomic status)</i>				
Fixed effects				
Individual level				
Social class‡ (reference = I–II)				
III nonmanual	1.19	0.91, 1.55	1.26	0.94, 1.69
III manual	1.00	0.75, 1.33	1.35	1.00, 1.83
IV–V	1.45	1.09, 1.94	1.45	1.07, 1.97
Unclassifiable	1.21	0.78, 1.89	1.24	0.80, 1.92
Mold or mildew in the home in the last year	1.12	0.89, 1.40	1.40	1.11, 1.78
Center level				
Percentage of low and medium education§	1.32	1.02, 1.72	1.44	1.12, 1.85
Random effects				
σ_u^2 (center)¶	0.098		0.077	
Median odds ratio	1.35#		1.30**	
<i>Model 2 (education and occupational exposure)</i>				
Fixed effects				
Individual level				
Educational level (reference = high)				
Medium	1.01	0.81, 1.26	1.20	0.93, 1.53
Low	0.92	0.64, 1.30	1.65	1.21, 2.26
High occupational exposure to biologic or mineral dust, gas, or fumes	1.09	0.80, 1.47	1.45	1.10, 1.92
Mold or mildew in the last year	1.10	0.88, 1.38	1.42	1.11, 1.79
Center level				
Percentage of low and medium education§	1.35	1.03, 1.78	1.32	1.02, 1.71
Random effects				
σ_u^2 (center)¶	0.105		0.073	
Median odds ratio	1.36††		1.29‡‡	

* Conveniently adjusted (multiple adjustment) for the variables shown in table 2, except sensitization and rhinitis.

† OR, odds ratio; CI, confidence interval.

‡ I, professional; II, managerial; III nonmanual, clerical; III manual, skilled manual; IV, semiskilled manual; V, unskilled manual.

§ Odds ratio for a difference of 28% (interquartile range) in the percentage of low and medium educational levels associated with the centers.

¶ Variance of the center-level random intercept.

Median odds ratio (MOR) of model with only age and sex = 1.46; MOR of model with only individual-level variables = 1.40.

** MOR of model with only age and sex = 1.44; MOR of model with only individual-level variables = 1.43.

†† MOR of model with only age and sex = 1.46; MOR of model with only individual level variables = 1.43.

‡‡ MOR of model with only age and sex = 1.44; MOR of model with only individual-level variables = 1.35.

categorized either by level of education or by social class group. The same was observed when atopics and nonatopics were assessed separately, although categorization by education showed no relation with asthma for atopics. We found

no evidence that these associations varied substantially between centers. Of interest, our study also indicated that regardless of their personal SES, subjects living in centers with a lower educational level had a higher risk of asthma.

This center-level association in part explains why some areas had a higher asthma prevalence than others.

Many possible risk factors for asthma are related to social class and have been proposed to explain associations between social class and asthma. We accounted for several of these risk factors including atopy, active and passive smoking, age, sex, body mass index, family size, birth order, infections in childhood, occupational exposures, and domestic mold growth, but the relation with socioeconomic indicators remained. Other important unmeasured components of SES could contribute to development of asthma. The role of early-life events such as maternal diet or the fetal and postnatal environment is one possibility (13). Childhood and adulthood obesity, inactivity, and diet have also been suggested as risk factors for asthma (32), but adjustment for body mass index did not confound the observed associations in our study. Environmental exposures outside the workplace, for example, air pollution, could be considered at the center level given the preliminary findings of an association between ozone levels, traffic-related ambient air pollution, and asthma incidence (33–35). Climatic variables such as temperature have also been suggested to be related to asthma (36), although its inclusion at the center level in our study did not modify the associations with social class and education (data not shown).

Identifying the specific role of each of these factors might be impossible if they exert their effect in early life or childhood. It has been hypothesized that differences in prevalence by SES are due to socioeconomic differences in exposure to allergens (12, 37), but we found that socioeconomic differences were present even after adjusting for sensitization to common allergens and also after stratifying by atopy. Other studies in poorer urban US communities (38) and in urban centers and suburban areas in Belgium (39) support our findings.

Another explanation for the socioeconomic differences could be that poor patients are more likely to have poorly controlled asthma (40–42), possibly because of less recognition of or concern regarding symptoms (43). The socioeconomic differences in management of asthma would be revealed by differential access to health services and/or differential prescription and/or use of asthma medication. Because provision of health care is universal in many of the countries in our study, access to health care is unlikely to explain the social class difference in asthma we observed. However, universal provision of medical care does equate with either equal access or equal utilization (43). In a previous study in the Spanish part of the ECRHS population, the frequency of seeing a physician was similar for symptomatic unemployed and employed subjects, but unemployed subjects were less likely to have been seen by a specialist (44). Such a difference seems unlikely to explain the entire association between asthma and low social class.

Regarding medication, some studies found that prescription rates of inhaled steroids (45) and the percentage of asthmatics receiving antiinflammatory drugs (32) were lower in lower socioeconomic groups. However, in our study, there were no differences by social class in the percentage of asthma medication or the percentage of inhaled and oral steroid use by those who reported asthma attacks (data not shown). Moreover, subjects in the low educational group

received more medication rather than less, as observed in previous studies (32).

Thus, poor treatment of asthma in the low socioeconomic groups is not likely to explain the increased asthma prevalence in these groups. The role of a variety of stressors and types of disadvantage such as violence or family disorder has been proposed to partially explain neighboring differences within a city (14). They also may account for center differences in our study, although we did not have this information.

It is important to note that the increased asthma prevalence in the lower SES categories was observed for both atopic and nonatopic subjects, even though atopy has been consistently related to high SES (12). Atopy did not modify the association between social class and asthma, low social class also being disadvantageous for atopic subjects, but atopics did not show differences by education. A recent survey in England found that high social class was negatively associated with wheezing in nonatopic subjects but was not associated with wheezing in atopic subjects (4). Differences in the effects of SES on asthma in atopic and nonatopic persons need further investigation.

Occupational exposure to dusts, gases, and fumes was associated with asthma in nonatopics, independent of educational level. Exposure to these nonspecific airborne agents in the workplace may aggravate a preexisting asthma condition or may well induce new-onset asthma following irritative mechanisms (46). The latter is relevant for nonatopic asthma rather than atopic asthma. However, findings in this cross-sectional analysis are probably influenced by health-related selection. It has been suggested that avoidance of occupational exposure by asthmatics with allergic occupational asthma has more benefits than it does for asthmatics with nonallergic occupational asthma (47). Mold or mildew in the home could be considered a marker of SES. It is associated with a higher risk of asthma in nonatopics. We speculate that mold exposure at the individual level is related to nonallergic asthma through airway inflammation due to nonviable fungal components (48).

A related issue concerns differential use of the term "asthma" by SES. The sensitivity of our definition of asthma compared with bronchial hyperresponsiveness was lower in the low education group (38 percent) than in the high education group (44 percent), and these differences were homogeneous among all centers, whereas sensitivity did not differ by social class (48 percent in the highest group vs. 51 percent in the lowest group). However, the observed differences in validity of the asthma diagnosis by education were of small magnitude and were unlikely to have biased our results. In addition, present findings remained unaltered when bronchial responsiveness was used as the indicator of asthma (data not shown).

At the center level, we found an association with educational level but not with social class. In large centers, mean social class may be a poor indicator of the community environment because of large within-center variations. On the other hand, education may be more homogenous because of within-center homogeneity of the educational system. Lacking information regarding small areas within a center precluded examination of poor inner-city areas, which have been reported to have disproportionately high asthma

morbidity in the United States (49). However, the effect at center level could be due to a country-level association, but there were not enough centers in some countries to build a three-level model to try to separate center-level from country-level variance.

Although in this cross-sectional study we could not examine whether low SES preceded the onset of asthma symptoms, there is no evidence that asthma in childhood reduces educational attainment. Among the strengths of this study were that data were gathered from multiple centers by using the same protocols and definitions, enabling us to apply multilevel models to assess heterogeneity between centers. In addition, we assessed an important set of potential confounders of the relation between asthma and SES. A possible limitation was the potential misclassification of asthma defined by questionnaire. However, the validity of the questions we used was good when compared with physician-diagnosed asthma (50). More refined classifications of social class have been proposed (51), but it is unlikely that applying them would have yielded significantly different results since they would not change the classification of most of the subjects in the low social class groups. We have to be cautious in interpreting associations at the center level since they were based on only 32 centers, and failure to demonstrate heterogeneity in the relations could be due to limited statistical power (52), although we exceeded the minimum number of groups suggested by some investigators: 30 (53).

In conclusion, in this large population representing several industrialized countries, SES was associated with asthma, with a higher prevalence among subjects of lower SES. Findings were consistent regardless of the SES indicator used. This association is, in part, explained by current and past individual exposures to lifestyle and environmental factors. Center educational levels were also related to asthma prevalence independently of individual-level education and social class.

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