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Occupational exposure to dust, gases and fumes, a family history of asthma and impaired respiratory health

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Objectives This study assessed the impact of occupational exposure to dust, gases, and fumes on respiratory symptoms, obstructive lung diseases, or the use of asthma medication among persons with and without a family history of asthma.

Methods A population-based cohort was followed for 10 years. This study included all 1739 men and 1594 women occupationally active at the first survey. Exposure and respiratory health were assessed from questionnaires. Multiple logistic regression was used to estimate the effects in relation to occupational groups, with age, gender, and smoking habits as possible confounders, using both cross-sectional and longitudinal analyses. The susceptibility to impaired respiratory health was determined from a family history of asthma.

Results A family history of asthma was reported by 27% of the men and 34% of the women. Both occupational exposure and a family history of asthma were associated with impaired respiratory health. The etiologic fractions showed that up to about 70% of the symptoms could be explained by a family history of asthma among those exposed to low levels of air pollutants, as well as among those with high exposure. However, high exposure contributed up to 35% of the symptoms both among those with and among those without a family history of asthma. The study indicates that the relative risk of occupational exposure to pollutants is similar for both persons with and those without a family history of asthma.

Conclusions The relative risk for impaired respiratory health after exposure to occupational air pollutants seems to be similar for persons with and those without a susceptibility to impaired respiratory health.

Key terms asthma; chronic bronchitis; disease susceptibility; epidemiology; etiologic fraction; respiratory symptom.

Several studies have found an association between occupational exposure to dust, gases, and fumes and respiratory health with respect to chronic cough, chronic phlegm, persistent wheeze, breathlessness, asthma, chronic bronchitis, or chronic obstructive pulmonary disease (1–10). It is well known that there are susceptibility factors for impaired respiratory health (eg, persons with a family history of asthma or atopy have an increased risk) (11). However, whether health effects of occupational exposure to air pollutants differ among persons with and those without susceptibility factors has been less studied (11). In this longitudinal population-based study, we estimated the impact of occupational

exposure on respiratory health in a general, adult, occupationally active population with respect to a family history of asthma.

Study population and methods

The study area was the northernmost county of Sweden, Norrbotten. The average annual temperature is approximately 0°C. The industrial setting in the area comprises heavy industries, including mining, steel production, forestry, and paper and pulp industries.

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The study was included in the OLIN (obstructive lung disease in northern Sweden) studies, details of which have previously been published. The study base included a stratified sample of men and women living in eight municipalities in Norrbotten in 1986. It started in the winter of 1986 with a prevalence study in which 86% of the invited persons responded to a postal questionnaire (12). The study was repeated in the winter of 1996 (13–15). Of those originally invited to participate in 1985–1986, 72% took part in both examinations. In these analyses, we have excluded all not occupationally active persons in 1986 (N=1521), such as students, housewives, old-age pensioners, early retirement pensioners, long-standing unemployed persons, and conscripts. Thereafter, the study sample comprised 1739 men and 1594 women born in 1934, 1935, 1949, or 1950.

Questionnaire

The questionnaire was based on that of the British Medical Research Council. It included questions about occupations, respiratory symptoms and diseases, the use of respiratory medication, smoking habits, and a family history of asthma (12). The questionnaire has been used in several studies in Baltic and Nordic countries (13, 16).

The following definitions and classifications have been used: (i) family history of asthma: a family history of asthma reported in 1986 or 1996; (ii) asthma-like symptoms: report of both attacks of shortness of breath and recurrent wheeze.

Prior and current occupations and the duration of each employment were requested in the questionnaire. Data on the occupational titles before 1986, in 1986, and in 1996 were used and coded according to the Nordic Classification System of Occupations (17). An experienced occupational hygienist (KE) recoded the occupational titles to those with high or low occupational exposure to dust, gases, and fumes.

In the cross-sectional study in 1986, the people highly exposed comprised those who, in or prior to 1986, had been highly exposed. The people with low exposure comprised those who, up to 1986, had always had low exposure. In the follow-up study in 1996, the people highly exposed comprised those who, in or prior to 1996, had been highly exposed. The people with low exposure comprised those who had always had a low level of exposure until 1996.

Analyses

Statistical analyses were performed using the Statistical Package for the Social Sciences for Windows, release 11.0.0 (SPSS for Windows, SPSS Inc, Chicago, IL,

USA, 2001) and SAS, version 8.12 (SAS Institute, Cary, NC, USA). A multiple logistic regression analysis was used to estimate the odds ratios. The analysis included smoking habits (nonsmokers, ex-smokers, smokers), age (two age groups), and gender as possible confounders. The fits of the models were described by log likelihood. Persons with missing data on occupational exposure, smoking, or a family history of asthma were excluded from the analysis. The analyses were repeated with the inclusion of only never smokers to study a possible residual confounding of smoking. A P-value of <5% was considered statistically significant.

Cumulative incidence was calculated as the proportion developing the studied condition between 1986 and 1996 among those who did not have the condition in 1986.

Cumulative remission was calculated as the ratio between those getting rid of the studied condition between 1986 and 1996 among those who had the condition in 1986.

The etiologic fraction (EF) was calculated as $EF = (OR - 1) / OR$, where OR is the odds ratio (18).

The ethics committee at the University and the University Hospital of Northern Sweden in Umeå approved the study.

Results

Basic characteristics

In 1986, there were 3333 occupationally active persons, of whom 1333 were classified as highly exposed (1008 men and 325 women, mean age 44 years), whereas 2000 were classified as having low exposure (731 men and 1269 women, mean age 43 years). Altogether 51 of the 3333 persons had stopped working before the 1996 survey, and 1309 persons had changed occupation between 1986 and 1996. The proportions with high exposure were 30% and 19% in 1986 and 1996, respectively. Before the 1986 survey, the participants had worked an average of at least 16 years. Between 1986 and 1996, 24% had changed exposure group. In 1986, there were slightly more smokers and ex-smokers in the highly exposed group. A total of 84% did not change smoking habits between 1986 and 1996 (table 1). A family history of asthma was reported by 27% of the men and 34% of the women.

Cross-sectional study in 1986

A high prevalence was found of chronic productive cough and physician-diagnosed chronic bronchitis for iron miners, of physician-diagnosed asthma and the use of asthma medication for workers in the paper and pulp

Table 1. Smoking habits in 1986 and 1986–1996 by exposure.

Exposure up to 1986	1986			Smoking habits 1986–1996							
	Non-smokers (%)	Ex-smokers (%)	Smokers (%)	Persistent nonsmokers (%)	Persistent ex-smokers (%)	Persistent smokers (%)	Starters (%)	Re-starters (%)	Quitters (%)	Unclear (%)	Unchanged smoking habits (%)
High	36	27	38	33	26	23	0.7	2.0	14	1.4	85
Low	44	23	33	41	23	22	0.6	2.1	11	1.0	82
Total	41	24	35	37	24	22	0.6	2.0	13	1.1	84

Table 2. Cross-sectional study in 1986—prevalence and odds ratios (OR) for combinations of a family history of asthma (FHA) and occupational exposure to dust, gases, and fumes in a multiple logistic regression with adjustment for age and gender. (exp = exposure, OR_E = odds ratio for occupational exposure, OR_F = odds ratio for a family history of asthma, OR_{EF} = odds ratio for the combined effect)

Dependant variable	Prevalence (%)				Model 1 ^a				Model 2 ^b							
	Low exp/ no FHA (N=1355)	High exp/ no FHA (N=936)	Low exp/ FHA (N=617)	High exp/ FHA (N=384)	OR _E	95% CI _E	OR _F	95% CI _F	Fit ^c	OR _E	95% CI _E	OR _F	95% CI _F	OR _{EF}	95% CI _{EF}	Fit ^c
Attacks of shortness of breath	6.6	8.8	15	21	1.6	1.2–2.0	2.5	2.0–3.2	2066	1.5	1.1–2.1	2.4	1.8–3.3	1.1	0.66–1.7	2066
Recurrent wheeze	7.2	12	16	24	1.5	1.2–1.9	2.4	1.9–3.0	2074	1.5	1.1–2.1	2.5	1.8–3.4	0.96	0.61–1.5	2073
Asthma-like symptoms	3.8	5.6	11	14	1.4	1.0–1.9	2.9	2.1–3.8	1405	1.5	0.95–2.2	3.0	2.0–4.6	0.88	0.49–1.6	1405
Longstanding cough	7.0	8.7	11	13	1.3	0.95–1.7	1.6	1.2–2.0	1873	1.3	0.95–1.8	1.6	1.2–2.3	0.97	0.58–1.6	1873
Sputum production	13	20	23	30	1.5	1.2–1.8	1.8	1.5–2.2	2764	1.6	1.2–2.0	1.9	1.5–2.5	0.86	0.58–1.3	2764
Chronic productive cough	2.3	3.4	4.2	4.0	1.3	0.81–1.9	1.5	1.0–2.3	877	1.5	0.88–2.6	1.8	1.1–3.1	0.64	0.28–1.5	876
Physician-diagnosed asthma	3.0	3.2	8.0	8.7	1.1	0.79–1.6	2.8	2.0–3.9	1190	1.1	0.67–1.8	2.7	1.8–4.2	1.1	0.55–2.1	1190
Use of asthma medication	2.7	3.2	9.1	8.1	1.2	0.82–1.7	3.1	2.2–4.3	1178	1.4	0.82–2.3	3.5	2.3–5.3	0.77	0.39–1.5	1178
Physician-diagnosed chronic bronchitis	2.1	3.6	3.3	7.1	1.9	1.2–2.8	1.8	1.2–2.6	908	1.7	0.97–2.8	1.5	0.86–2.7	1.3	0.60–2.9	908

^a Includes dummy variables for exposure and FHA.^b Includes dummy variables for exposure and FHA, and the combined effect.^c Estimated by –2 log likelihood.

industries, and of chronic productive cough for farmers (data not shown).

In the multivariate analysis, respiratory health was impaired by both occupational exposure and family history of asthma (table 2). An analysis restricted only to nonsmokers showed similar results, except for physician-diagnosed chronic bronchitis, for which there were no increased risks (data not shown). The odds ratios for the combined effect of a family history of asthma and exposure were not significant, and the fit of the model did not increase significantly. This finding indicates that the relative risks of exposure were similar for the persons with and those without a family history of asthma (table 2).

Longitudinal study, 1986 to 1996

Incidence. The cumulative incidences increased more among those with high exposure and with a family history of asthma, showing a pattern similar to that of the

cross-sectional analyses (table 3). The analysis showed increased risks for all symptoms and diagnoses among the exposed persons; however, the odd ratios were only significantly increased for some symptoms. The analysis did not show any better fit if the second model and the odds ratios for the combined effect of a family history of asthma and exposure were not significant. An analysis of only nonsmokers showed similar results (data not shown).

Risk factors other than occupational exposure. The odds ratios for other major risk factors used in the model, such as age, gender, a family history of asthma, and smoking habits, were not very different from what we have recently published in a similar study of socioeconomic status based on the same material (no new data shown) (19).

Etiologic fraction. When the odds ratios were transformed to etiologic fractions, it was obvious that a fam-

Table 3. Longitudinal study from 1986 to 1996—cumulative incidence and odds ratios (OR) for combinations of without or with a family history of asthma (FHA) and occupations with high or low exposure to dust, gases, and fumes in a multiple logistic regression with adjustment for age, smoking, and gender. (OR_E = odds ratio for occupational exposure, OR_F = odds ratio for a family history of asthma, OR_{EF} = odds ratio for the combined effect)

Dependant variable	Cumulative incidence (%)				Model 1 ^a					Model 2 ^b						
	Low exp/ no FHA (N=1355)	High exp/ no FHA (N=936)	Low exp/ FHA (N=617)	High exp/ FHA (N=384)	OR _E	95% CI _E	OR _F	95% CI _F	Fit ^c	OR _E	95% CI _E	OR _F	95% CI _F	OR _{EF}	95% CI _F	Fit ^c
Attacks of shortness of breath	7.9	8.7	19	22	1.3	1.0–1.7	2.8	2.2–3.5	1904	1.3	0.94–1.8	2.8	2.0–3.8	1.0	0.63–1.7	1904
Recurrent wheeze	6.5	8.3	13	19	1.4	1.0–1.9	2.3	1.7–3.0	1486	1.3	0.90–1.9	2.1	1.4–3.1	1.2	0.69–2.1	1485
Asthma-like symptoms	4.5	6.1	12	18	1.6	1.2–2.2	2.9	2.2–3.9	1306	1.5	0.97–2.3	2.7	1.8–4.2	1.2	0.64–2.1	1306
Longstanding cough	4.1	6.6	7.5	8.4	1.1	0.87–1.5	2.1	1.6–2.7	1732	1.3	0.94–1.9	2.4	1.7–3.4	0.70	0.42–1.2	1730
Sputum production	9.3	12	14	20	1.3	0.96–1.7	1.7	1.3–2.3	1654	1.2	0.86–1.7	1.6	1.1–2.3	1.2	0.68–2.0	1654
Chronic productive cough	4.8	6.6	7.5	8.4	1.3	0.95–1.9	1.6	1.2–2.2	1322	1.6	1.0–2.4	2.0	1.3–3.0	0.65	0.35–1.2	1320
Physician-diagnosed asthma	3.5	3.4	11	14	1.3	0.95–1.9	3.6	2.6–5.0	1275	1.1	0.70–1.9	3.2	2.1–4.9	1.3	0.71–2.5	1274
Use of asthma medication	6.7	6.4	17	16	1.1	0.80–1.4	2.7	2.1–3.5	1810	1.1	0.75–1.6	2.8	2.0–3.9	0.94	0.57–1.6	1810

^a Includes dummy variables for exposure and FHA.

^b Includes dummy variables for exposure and FHA, and the combined effect.

^c Estimated by –2 log likelihood.

ily history of asthma explained more of the occurrence of these symptoms than occupational exposure in this population. A relative risk of up to 3.6 indicated that up to about 70% of the symptoms could be explained by a family history of asthma, both for those with low exposure and those with high exposure. A relative risk of 1.6 indicated that high exposure contributed up to about 35% of the symptoms.

Remission. Cumulative remission was lowest for physician-diagnosed asthma and the use of asthma medication, 10% and 14%, respectively. Otherwise the median was 37%, and the range was 21–72%, the highest value occurring for chronic productive cough. The number of incident cases was found for physician-diagnosed asthma and the use of asthma medication, 12 and 14 times higher, respectively, than for the remittent cases. For other symptoms, the corresponding figures were lower, the median being 2.5 and the range being 1.2–4.6.

Discussion

This population-based cohort study of occupationally active workers showed that respiratory health is impaired by occupational exposure to dust, gases, and fumes among persons with and those without a family history of asthma.

Our findings of an association between occupational exposure and respiratory health are in accordance with

the results of other studies (1–10). Other population-based cross-sectional studies have found odds ratios of 1.8 to 3.2 for asthma after exposure to dust, gases, and fumes (4, 5), and, in one of them, an exposure–response relationship was found (5). Results based on longitudinal studies are still few. A study from Norway found an increased risk of 1.6 for developing asthma among workers exposed to dust, gases, and fumes (9). A Finnish study based on the Medication Reimbursement Register of the Social Insurance Institution of Finland, the Finnish Register of Occupational Diseases, and Finnish population census data from 1985, 1990, and 1995 found an increased incidence of asthma especially in manufacturing, service, and agricultural work, ranging from 1.53 to 2.12 for men and from 1.33 to 1.84 for women when administrative work was used as the reference group (8).

Estimating exposure from occupational titles is a crude method. Our characterization of exposure does not take into account either the cumulative exposure or the very different kinds of exposures. As the classification of exposure was blinded according to outcome, there is a low risk of differential bias due to the classification of exposure. A nondifferential misclassification of exposure tends to decrease the risk estimates. Other population studies have, as we have, used occupational titles for estimating exposure (3). Some studies have used data from interviews (1) or persons' self-reported exposures (4, 9). Both when and for how long a time people have been exposed vary in different studies. We have classified a person as highly exposed if he or she has ever worked

in a highly exposed job, a method used by other authors (4, 9). Other studies have based their results on the last occupation (7, 20), the longest-held occupation (21), or the occupation during the first 5 years of a follow-up (2). The choice of method may affect the results. When we ignored exposure from occupations held before the survey in 1986, the effects of exposure were somewhat weaker. This finding indicates that also exposure for a long time before the survey may have had effects. This finding is also in accordance with our previous results, in which retired miners had an increased prevalence of respiratory symptoms (22) and respiratory symptoms were more common among ex-smokers than among nonsmokers (23).

As expected, we found a family history of asthma to be a strong determinant of respiratory health, especially for asthma, and this finding agrees with the results of other studies (13, 15, 19, 22, 24). As people with a family history of asthma have a higher risk of impaired respiratory health a priori, the risk in absolute terms is higher among the susceptible persons. We do not believe our results should be interpreted as a cause for counseling susceptible persons to avoid working in environments with high exposure to dust, gases, or irritants. Rather it shows that preventive measures to reduce exposure are beneficial both for susceptible and nonsusceptible persons. On the other hand, the environment as a possible cause of impaired respiratory health should not be neglected among persons with a susceptibility for airway disease, and reducing the exposure should be one option in the therapeutic arsenal.

The studied variables can be classified as asthmatic variables (attacks of shortness of breath, recurrent wheeze, asthma-like symptoms, physician-diagnosed asthma, and the use of asthma medication) and as bronchitic variables (longstanding cough, sputum production, chronic productive cough, and physician-diagnosed chronic bronchitis). The symptoms often overlap. Bronchitic symptoms are strongly associated with smoking. We had no data on pack-years, and therefore the smoking data (classified as never smoker, ex-smoker, and smoker) was used in the adjusted analyses. To study possible unadjusted confounding for smoking, we repeated the analysis for nonsmokers only. The power was much lower, but the findings indicated that the possible unadjusted confounding of smoking was weak.

Risks can be expressed as odds ratios or etiologic fractions. An etiologic fraction indicates how much of a condition could be prevented if the determinant could be eliminated (18). For attacks of shortness of breath and recurrent wheeze, etiologic fractions of about 15% have been reported in cross-sectional studies (21, 25). In a Norwegian longitudinal study these figures were about 5% to 10%, and for sputum production, chronic productive cough, and physician-diagnosed asthma they were

about 15% (9). A Finnish longitudinal population study found 29% for men and 17% for women (8). When the wide confidence intervals are considered, our findings are similar in size.

In summary, the study shows that occupational exposure to dust, gases, and fumes is a determinant of respiratory health both for persons with and for those without the susceptibility factor of a family history of asthma.

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