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Bronchial asthma and air pollution at workplaces

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Objectives This case-referent study was performed to investigate the possibility of nonspecific air pollution at workplaces increasing the risk of bronchial asthma for formerly healthy adults.

Methods Seventy-nine cases of asthma, diagnosed at a lung clinic, among persons aged 20—65 years were compared with 304 referents drawn randomly from the population of the catchment area. Questionnaire information was obtained regarding occupation, exposure to suspect allergens, place of residence, smoking habits, and atopy. The subjects' occupations were categorized into four air pollution classes based on how the referents reported air pollution in their respective occupations.

Results Three years or more of work in air-polluted occupations resulted in an odds ratio of 3.0 (OR) (95% confidence interval 1.5—6.1) in a comparison with work in occupations with slight or no air pollution. Stratification of the material for smoking habits or atopy did not alter the results. Nor did the exclusion of specific exposures to asthmagenic agents such as isocyanates, stainless steel welding, or aluminum salts change the effects of the nonspecific air pollution at workplaces. Smoking per se was associated with an almost doubled risk for asthma (OR 1.9, 95% confidence interval 1.1—3.4).

Conclusion The results of this study support an association between occupational exposure to nonspecific air pollution and the development of bronchial asthma.

Key terms case-referent study, occupational exposure.

The role of nonspecific air pollution at workplaces in the development of bronchial asthma (hereafter referred to as "asthma") is unclear. From our clinical experience, asthma is more common among workers in occupations with exposure to dust, smoke, and vapors. Little information was available in the literature about any connection between nonspecific air pollution at work and asthma when we started this study. However, in recent years, some indications of such an association have been obtained. In a case-referent study from Singapore the odds ratio for asthma was significantly increased for manual workers and service personnel, when compared with non-manual workers, after adjustment for smoking and other factors (1). Similar results were obtained in a community-based cross-sectional study from China, where a relationship between occupational exposure to dust, gas, or fumes and physician-diagnosed asthma has been reported (2). In a Norwegian cross-sectional study of the general population, a history of occupational exposure to dust and gas was also associated with physicians' diagnoses of asthma (3).

Contrary to the sparse literature on the effect on nonspecific air pollution, the literature abounds with reports on the effect from specific occupational exposures, for example, to isocyanates, some kinds of tropical wood, molds, and animals (4).

The objective of the present study was to assess the risk of a gainfully employed population developing asthma from occupational exposure to nonspecific air pollution.

Subjects and methods

Cases of asthma were collected from two adjacent rural communities, with altogether 46 000 inhabitants and situated in the southeast of Sweden. The wood industry is dominant in the area, for example, with the production of prefabricated wooden houses and furniture.

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pharmacies of the two communities all prescriptions of beta agonists, used mainly by asthmatics, were collected during approximately a nine-month period in 1990. Altogether 271 persons who had received beta agonist prescriptions were identified. The physicians responsible for the prescriptions were contacted by letter and asked to confirm or refute the asthma diagnosis for each patient who had a prescription. As a result 89 persons were excluded as suffering from bronchitis, emphysema or some other, nonasthmatic disorder. After this first verification of the diagnosis, 182 subjects remained as possible asthmatics. We then contacted these persons with an introductory letter that preceded a questionnaire which was answered by 167 subjects (92%).

The diagnostic criteria for asthma that we ultimately applied were those suggested by the American Thoracic Society (5), namely, (i) a clinical history of reversible bronchial obstruction and (ii) either pathological results in a methacholine test [ie, at least a 20% decrease in forced expiratory volume in 1 s ($FEV_{1.0}$)] after the inhalation of methacholine in increasing concentrations from 0.125 to 8 mg · ml⁻¹, or (iii) significant results in a reversibility test, (ie, inhalation of beta-2-agonists resulting in at least a 15% increase in $FEV_{1.0}$). We excluded those who did not fulfill these criteria, as determined by a lung specialist. The result was an additional exclusion of 44 subjects. Since we focused on adulthood asthma, we also excluded cases with onset of disease before the age of 20 years, which meant a loss of another 44 persons. Thus 79 asthmatics who were aged 20–65 years at diagnosis remained. One of us (JZ) examined all the persons at the same lung clinic.

The mean age at diagnosis for the 79 asthmatics was 39 years. For the 304 referents the mean age was 37 years at the time their matched cases were diagnosed with asthma. Fifty-seven percent of the cases and fifty-eight percent of the referents were men.

The referents, aged 20–65 years in 1990, were randomly drawn from the general population register of the catchment area for the cases through the National Health Insurance. For each of the originally identified potential cases, three referents were primarily selected, all in all 539 persons. They received an introductory letter followed by the questionnaire one week later. Four hundred and seventy-two referents answered the questionnaire (88%).

Since the cases had been diagnosed in 1950–1989 and we were interested in exposures prior to the onset of asthma, taken as the year of diagnosis, an anchor point in time had to be found also for the referents. For this purpose four referents from the pool of 472 referents were matched to each of the 79 cases by gender, municipality, and age. This procedure permitted exposure assessment of these referents in relation to the year of diagnosis of the corresponding case. We accepted an

average age difference of four years within an age range of 0–11 years between a case and its matched referents. Three-hundred and four referents fulfilled the matching criteria, whereas 12 did not and were excluded.

Assessment of exposure

Information on exposure was obtained with a 10-page questionnaire regarding workplace conditions, including job titles, work tasks, and the occurrence of dust, fumes, vapors and passive smoking. Specific exposure to animals and some 20 different work tasks were inquired about. Questions on smoking habits, moisture damage and close-fitted carpets in dwellings, allergy in childhood, and some other medical issues were also included.

We classified the job titles into categories of increasing probability of exposure to air pollution by using the answers of the referents on the questions about dust, fumes, and vapors reported in connection with the various job titles.

A work task was classified as clean when only 0–33% of the referents classified it as involving exposure to dust, fumes, or vapors. This group formed the reference or null category. If 34–50% of the referents holding a job title reported their job as air polluted, the job title was referred to the low-exposure category, that is, category 1. When 51–75% complained, the jobs were placed in category 2, and, finally, if over 75% of the referents complained, the job title was classified as highly exposed, that is, category 3.

It should thus be noted that the exposure categories obtained indicate the probability of exposure to air pollution at work and not exposure levels per se. The following occupations belonged to category 3: librarian, farmer, gardener, railway engineer, post-office worker, spinner, weaver, upholsterer, seamstress, leathersewer, roller, caster, watchmaker, dental technician, plater: tinsmith, welder, electrical installer, telegraph repairman, workshop carpenter, mason, building trade: wood worker, other wood worker, bookbinder, chemical process industry worker, rubber industry worker, paper pulp worker, concrete worker, water supply engine operator, construction machine operator, janitor, dentist, forwarding agent, well driller, miner, trade ship officer, engine operator, shoemaker, furrier, goldsmith, metal plater, machine electrician, woodfiber worker, boat builder, painter, glazier, glass blower, engraver, mill worker, injection molding, machine worker, fire brigadier, chimney sweeper, policemen, purser, laundry presser and trotting coach.

Since we were interested in testing the hypothesis that nonspecified air pollution at work could be a risk factor for bronchial asthma, we merged the exposure categories for dust, fumes, and vapors for each subject, so as to obtain one occupational air-pollution category for each person. The highest exposure category for either

dust, fumes, or vapors determined the occupational air-pollution category that the subject was placed in. Thus a subject belonging to, for example, dust category 2, fume category 1, and vapor category 0 was regarded as belonging to occupational air pollution category 2. The exposure categories were merged in this manner because our material was fairly small. Attempts to present odds ratios for single exposures or occupations would have led to very small numbers and to risk estimates that would be too unstable.

Epidemiologic analyses

The year of onset of asthma was taken as the year of diagnosis given by the responsible physician. A minimum exposure requirement of three years, all before the year of diagnosis, was applied for all types of exposures, also ever smoking. We calculated the crude odds ratios for all single exposures that had lasted for three years or more. No lag time demand from the end of exposure until the year of diagnosis was used, except for ex-smoking, which meant that smoking had ended at least one year before the diagnosis of asthma; otherwise the subject was considered a smoker. Confounding in the data was identified by considering the correlation between the risk indicators among the referents (6).

The final analyses were performed by using the EGRET package for multiple logistic regression (7) and adjusted for occupational animal contact, allergy in childhood, passive smoking at work, ever smoking, age at diagnosis, and gender.

Results

In the crude analyses, exposure category 3 for both dust and fumes, as well as occupational air-pollution category 3, were significant risk indicators (see table 1). Occupational contact to animals and ever smoking also increased the risk significantly. Passive smoking at work brought a slight but nonsignificant risk, whereas passive smoking at home did not.

Stratifying the material on allergic symptoms in childhood, age at diagnosis, decade of onset of asthma and gender resulted in a numerically different risk between strata from occupational air-pollution category 3 although the 95% confidence intervals (95% CI) overlapped (table 2). The risks after stratification for smoking were similar.

To ensure that exposure to air-polluted work did not merely aggravate existing asthma and cause a false association between exposure and asthma, we also performed a supplementary analysis in which we introduced a lag time of one year. Hence exposure within one year before the diagnosis was disregarded. Three years or more of

exposure to air-polluted work resulted in an odds ratio of (OR) 3.1 (95% CI 1.6–6.0), which is similar to the result when no lag time was used (not shown in table).

The exclusion of subjects with exposures or jobs with exposure to established asthma-causing agents (7 cases and 30 referents) (ie, aluminum salts, soldering, isocyanates, bakers, chimney sweepers, hairdressers, foundry and rubber factory workers), which were specifically

Table 1. Exposure or state of the subjects with bronchial asthma and of the referents, along with the crude odds ratios. (OR = odds ratio, 95% CI = 95% confidence interval)

Exposure/state	Number		OR	95% CI
	Cases (N=79)	Referents (N=304)		
Occupational animal contact	21	47	2.0	1.0–3.7
Ever smoking	50	145	1.9	1.1–3.3
Occupational dust exposure category 3 versus 0	36	94	2.5	1.3–4.8
Occupational fume exposure category 3 versus 0	8	6	5.8	1.6–21
Occupational vapor exposure category 3 versus 0	2	4	2.3	0.2–17
Occupational air pollution category ^a				
0	18	133	(1.0)	—
1	9	26	2.5	0.9–6.8
2	10	49	1.5	0.5–3.7
3	42	96	3.2	1.6–6.3
Passive smoking				
At work	35	104	1.5	0.8–2.6
At home	26	108	0.9	0.5–1.5
Allergy symptoms in childhood	14	40	1.4	0.6–2.9

^a The occupational air pollution category was constructed by merging exposure to dust, fumes and vapors. See the section on exposure assessment in the text.

Table 2. Odds ratios (OR) for exposure to air pollution category 3 versus air pollution category 0 among the cases with bronchial asthma and the referents in relation to ever smoking, allergy in childhood, gender, age at diagnosis, and decade of diagnosis. (95% CI = 95% confidence interval)

	Exposed-cases	Exposed referents	OR	95% CI
Ever smoking				
No	17	45	3.2	1.2–9
Yes	25	51	3.0	1.2–8
Allergy in childhood				
No	36	85	3.6	1.7–7.9
Yes	6	11	2.2	0.4–11.7
Men	33	69	5.0	1.7–17.6
Women	9	27	2.0	0.6–5.8
Age at diagnosis				
20–34 years	20	36	7.0	2.4–23
35–49 years	14	39	1.6	0.5–4.6
50–65 years	8	21	2.1	0.4–14.4
Year of diagnosis				
1950–1959	2	2	4.2	0.1–353
1960–1969	5	16	2.3	0.3–28
1970–1979	8	12	5.6	1.2–30
1980–1989	27	66	2.9	1.2–7

Table 3. Logistic odds ratios for exposures of interest among the cases with bronchial asthma and the referents.^{a,b} (95% CI = 95% confidence interval)

Exposure/state	Log odds ratio	95% CI
Occupational air pollution category		
1 versus 0	2.6	1.0—6.6
2 versus 0	1.5	0.6—3.8
3 versus 0	3.0	1.5—6.1
Occupational animal contact	1.8	0.8—3.4
Allergic symptoms in childhood	1.8	0.8—3.6
Passive smoking at work	1.5	0.8—2.5
Ever smoking	1.9	1.1—3.4

^a According to logit $f(y)$ case referent = 4.254 + 0.9647 (occupational air pollution category 1 versus 0) + 0.4275 (occupational air pollution category 2 versus 0) + 1.110 (occupational air pollution category 3 versus 0) + 0.5628 (occupational animal contact) + 0.5748 (allergy in childhood) + 0.3745 (passive smoking at work) + 0.1872 (age 35—49 years at diagnosis) + 0.2007 (age 50—65 years at diagnosis) + 0.6595 (ever smoking) — 0.4614 (female gender).

^b The occupational air pollution category was constructed by merging exposure to dust, fumes and vapors. See the section on exposure assessment in the text.

asked for in our questionnaire, did not change the increase in risk for the highest air-pollution category (OR 3.2, not shown in table).

Considering the correlation between different exposures, we found that occupational air-pollution category 3 was connected with other risk factors also, thereby indicating confounding. Therefore multiple logistic regression was utilized for the final analysis. As a result, "occupational air pollution" categories 1 and 3 and smoking remained as the strongest risk factors, but there was no clear trend (table 3).

Discussion

Methodology

Asthma cases were recruited through the prescription registers of the all the pharmacies of two municipalities. To meet our aims of catching all asthmatics of the involved municipalities, the cases should ideally have bought their medicine at the pharmacies of their home community. Extensive commuting to other communities, where the asthmatics could hypothetically buy their medication, would have distorted the case collection. However, only about 10% of the working population daily commuted to other communities (national census 1990).

The diagnosis of asthma is difficult. To ensure the best possible diagnostic validity and to distinguish asthma from chronic obstructive pulmonary disease, we therefore included a lung specialist (JZ) in our group and applied the diagnostic criteria suggested by the American Thoracic Society (5). To our knowledge, these criteria are the best available for practical purposes, and superior to those often used in previous epidemiologic studies, for example, on smoking and asthma (8).

The referents were randomly selected from the general population register through the National Health Insurance. They thus represented all adult inhabitants of the two municipalities without selection bias. The response rate was satisfactorily high, 92% for the cases and 88% for the referents.

The occupations were classified into categories of air pollution according to the opinions of the referents. Thereby observation bias of exposure by the authors or by the asthmatics should have been eliminated. A few occupations classified by the referents as air polluted, as indicated in the section on the assessment of exposure, might seem strange, for example, librarian or dentist. However, the consequence of including some persons who might actually have low exposure into the high-exposure category would lead to a weakening of the association between exposure and disorder rather than to an increased risk.

Comment on the results

The main result in this study was that occupational exposure to nonspecified air pollution for more than three years causes an increased risk of developing asthma. This result is in agreement with that of two recently published papers (1, 2). Ng et al (1) found an odds ratio of 1.7 for occupations related to service and production in a comparison with an unexposed reference group of administrative, managerial, clerical, and sales occupations in a community-based case-referent study. Xu & Christiani (2) performed a community-based prevalence study on physician-diagnosed asthma and reported an odds ratio of 1.6 for groups exposed to dust and gas or fumes. An exposure-response relationship for cumulative exposure — a combination of duration and intensity — was reported for gas or fumes and dust with odds ratios of about 2. Xu et al (2) stated that their study was the first to demonstrate a relationship between occupational exposure to air pollution and physician-diagnosed asthma. Already in 1991, however, Bakke et al (3), in a cross-sectional study, found an odds ratio of 1.7 (95% CI, 1.2—2.5) for asthma for subjects exposed to dust or gas. Antti-Poika et al (9) found no correlation between long-term exposure to airway irritants at work and asthma in twins. The authors noted however that a drawback of the design of their study is the use of twins, who usually tend to choose similar occupations. Besides, a cross-sectional approach also reduces the probability of finding an effect of exposure, since asthmatics, because of their bronchial hyperreactivity, would be prone to leave a dusty occupation and choose a clean job.

Torén et al (10), in a register-based nationwide cohort study from Sweden, observed increased mortality among workers in some polluted, and also some clean, jobs. Since no information was obtained about the start of exposure and year of onset of asthma, the results are

difficult to interpret as to the role of an asthmatogenic effect of air pollution at workplaces.

The odds ratio in our study is somewhat higher than found in the aforementioned papers (1—3). It may be that our exclusion of asthmatics with onset in childhood is one reason. Including them would have raised the background risk and therefore obscured the effects of exposures as adults. Furthermore, we also considered only exposure prior to a diagnosis of asthma to avoid the selection effects of disease on exposure, which might also have led to a lowered risk estimate.

The risk ratio for asthma among smokers exposed to air pollution at work was similar to that of exposed nonsmokers. Therefore a multiplicative effect of the two exposures combined is indicated. This finding is somewhat in agreement with the finding of higher risk for allergic asthma among smoking workers exposed to tetrachlorophthalic anhydride (TCPA) or isphagula and among coffee workers and crab processors (11). However, the lower risk for asthma among atopic subjects exposed to air pollution in our material suggests an irritative rather than an allergic mechanism (12).

Despite the generally low levels of air pollution at workplaces in the past decade in Sweden, the current hygienic standard does not seem to be good enough to protect for asthma induction, since also air-polluted workplaces in the 1980s seemed to involve a risk (table 2). Thus there still seems to be room for improving the environment at many workplaces in Sweden, even if the hygienic standard might be thought of as good from an international perspective.

The risk for asthma in air-polluted workplaces was numerically higher for subjects between the ages of 20 and 34 years. Therefore it may be that only a short exposure time is needed for inducing asthma, and susceptible persons fall ill already at young ages. It is also likely that the background occurrence of asthma is higher at older ages and thereby reduces the odds ratio.

We have no explanation for the greater risk for men than for women for asthma at air-polluted workplaces. For children the susceptibility to air pollution is reportedly greater among girls than boys. Forastiere et al (13) reported a greater frequency of response to methacholine for girls when living in an area polluted from sulfur dioxide and total suspended particulates. From Sweden, similar results have been found for children exposed to automobile exhaust monitored as nitrogen dioxide, which brings girls a greater risk of wheezing bronchitis, a disorder that means an increased risk for asthma (14).

The mechanisms for developing asthma after long-time exposure to air pollution are unclear. Possibly, a chronic inflammatory reaction of the mucous membranes results from the air pollution, as suggested in some case reports after short-time exposure to high concentrations

of irritating agents like ozone, chlorine and phosgene (15), hydrogen chloride (16), and sulfuric acid (17), as well as nonspecific air pollution from fire smoke (15). Irritant-induced occupational asthma is a concept used by Tarlo & Broder (16) while Brooks et al (18) have coined the term "reactive airway dysfunction syndrome," including airway hyperreactivity as a result of short-time exposure to the aforementioned chemicals.

The association between air-polluted jobs and asthma found in our study might include exposure to specific allergens, although not identified except for the 7 cases and 30 referents who were excluded from the analysis of this material. For farm work both specific allergens like mites and dander and nonspecific dust might be responsible for the increased asthma risk reported in the literature (10). In a Swedish study 25% of asthmatic farmers had antibodies against mites and another few percent had antibodies against dander (19). Overall, about one-third of the farmers were sensitized (personal communication, Rask-Andersen 1996). For the majority of asthmatic farmers in that study, no allergic signs were apparent. Wood workers in Sweden are mainly exposed to soft wood-like fir and pine, which are not known to be sensitizing. It cannot be excluded, however, that some wood workers are exposed to tropical species of wood. Welders in Sweden mostly work on mild steel. Only a minority — about 13% of Swedish welders — work on stainless steel according to a survey in 1974 (20). Furthermore, in another Swedish study, the incidence of asthma among stainless steel welders was slightly lower than among mild steel welders (21).

Painters can be exposed to diisocyanates, which are specific sensitizers. It was possible to exclude subjects exposed to diisocyanates from the analysis since we asked for this exposure in the questionnaire. Some might have escaped the exclusion, however, if their exposures were not reported properly. Regarding other jobs with potential risks for sensitizing exposures, it is noteworthy that only referents and no cases in our material were dentists or dental technicians, with potential exposure to methyl methacrylate, or metal platers, with potential exposure to chromium and nickel.

In conclusion, and regarding specific sensitizing or nonspecific irritating agents causing asthma in our material, we consider the excess of asthma observed in our material as mainly being a result of nonspecific exposures. In a self-administered questionnaire on exposure, it is difficult to get precise information, however, and therefore further studies would benefit from a more thorough description of the workplaces by occupational hygienists.

Another result of our study was that active smoking doubled the risk for asthma. This aspect has been presented in a previous paper (8), where also the effect of passive smoking at work is discussed.

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