Occupational Exposures and Asthma in 14,000 Adults from the General Population

Nicole Le Moual1, Susan M. Kennedy2, and Francine Kauffmann1

1 Epidemiology and Biostatistics, INSERM U472, Villejuif, France.
2 University of British Columbia, Vancouver, British Columbia, Canada.

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The association of asthma with occupational exposures was studied in 14,151 adults, aged 25–59 years, from the general population of the 1975 French Pollution Atmosphérique et Affections Respiratoires Chroniques (PAARC) Survey. Associations of asthma with specific jobs, such as personal care workers, waiters, and stock clerks, were observed, with age-, sex-, and smoking-adjusted odds ratios between 1.5 and 1.7. Exposures to 18 asthmagenic agents (low and high molecular weight and mixed environment) were estimated by an asthma-specific job exposure matrix. Risks associated with asthma increased when subjects with imprecise estimates of exposure were excluded. Risks increased further with increasing specificity of the definition of asthma when considering jobs or specific agents, such as industrial cleaning agents, latex, flour, highly reactive chemicals, and textiles. For example, for industrial cleaning agents, odds ratios increased from 1.55 (95% confidence interval (CI): 1.08, 2.23) for “ever asthma,” to 2.17 (95% CI: 1.41, 3.34) for asthma onset after age 14 years, to 2.35 (95% CI: 1.38, 4.00) for asthma onset after beginning current job, and to 2.51 (95% CI: 1.33, 4.75) for asthma with airflow limitation. Results underlined the importance of the specificity of exposure and asthma definitions and indicated a deleterious role of occupational exposure on asthma, especially for cleaning agents.

asthma; occupational diseases; occupational exposure; occupations

Abbreviations: CI, confidence interval; ECRHS, European Community Respiratory Health Survey; EGEA, Epidemiologic Study of the Genetics and Environment of Asthma; FEV1, forced expiratory volume in 1 second; ISCO88, International Standard Classification of Occupations, 1988; OR, odds ratio; PAARC, Pollution Atmosphérique et Affections Respiratoires Chroniques.
gases) (13) and a second that estimated exposures to specific asthmagenic agents (14).

In previous analyses of the French Pollution Atmosphérique et Affections Respiratoires Chroniques (PAARC) Survey, five estimates of exposure (self-reported exposure (17–20), three cancer-specific job exposure matrices (18, 20, 21), and a population-specific job exposure matrix (19)) were used to study the associations between occupational exposure and asthma or lung function. Positive associations between self-reported exposure to dusts, gases, and fumes and asthma were observed, whereas no association was found when exposure was estimated by a job exposure matrix (18). Our objective was to investigate, in the PAARC Survey, the associations between occupational exposure (estimated using four methods suitable for large population studies) and asthma (defined in four ways) in an attempt to examine the role of increasing specificity of both definitions in the associations seen.

MATERIALS AND METHODS

Population

The PAARC Survey was carried out in 1975 in seven French cities, on 20,310 subjects, aged 25–59 years, surveyed at home. More detailed protocol has been published elsewhere (17, 22). The primary aim was to investigate the effect of air pollution on respiratory outcomes; thus, households headed by manual industrial workers (mostly men) were excluded to reduce the effect of occupational exposure. Other manual workers, such as craftsmen, were included.

Smoking habits and asthma

The questionnaire was based on the British Medical Research Council/European Coal and Steel Community questionnaire. Subjects were classified in five categories as nonsmokers, former smokers, and light, moderate, and heavy smokers on the basis of grams of tobacco smoked per day. Questions regarding asthma were “Have you ever had attacks of breathlessness with wheezing?” and “Have you ever had asthma attacks?” and, if yes to one of the previous questions, subjects were asked for the “age at the first attack” and the “age at the last attack.” For this analysis, “ever asthma” was defined as a positive response to at least one of the first two questions. Additional definitions were used to characterize more specific phenotypes. A surrogate for severe asthma was estimated by combining a positive report of asthma symptoms with airflow limitation data, as no other markers of severity were available in the PAARC Survey. Thus, “asthma with airflow limitation” was defined as “ever asthma” plus forced expiratory volume in 1 second (FEV1)/forced vital capacity of less than 88 percent of the predicted value in men and 89 percent in women, based on the European reference values (23). Two specific definitions of “adult-onset asthma” were used: asthma onset at or after age 14 years and asthma onset after beginning the current job. Age 14 years was chosen because it corresponded, for many people born between 1916 and 1950 (i.e., the PAARC Survey generation), with the age of beginning work or apprenticeship.

Estimates of exposure

Four estimates of exposure were used and summarized in figure 1. First, self-reported exposure to dusts, gases, and fumes in the current or most recent occupation was estimated by the response to this question: “Were you exposed to dusts, gases, or chemical fumes?” Second, job titles, coded using national French codes, were grouped into 29 categories based on categorization schemes previously used by other authors (24, 25) and compared with a reference group of administrative and service jobs. Third, a population-specific job exposure matrix for “dusts, gases, and fumes” was constructed using self-reported exposure and job codes as previously described (19). Briefly, each job code was classified as either “exposed to dusts, gases, and fumes” or “not exposed,” on the basis of the proportion of subjects holding that job who declared themselves exposed. The exposure is estimated by job codes and then ascribed to all subjects with this job. The cutoff proportion to distinguish exposed and not exposed jobs was selected a priori to ensure that the overall percentage of persons classified as exposed to dusts, gases, and fumes would be equal whether one used the job exposure matrix or self-report (figure 1). Fourth, an asthma-specific job exposure matrix (14) was used to estimate exposure to asthmagenic agents after translation of French codes (26) into International Standard Classification of Occupations, 1988 (ISCO88), codes (27). This asthma job exposure matrix, which assigns exposure estimates for all ISCO88 job codes, was developed for the Epidemiologic Study of the Genetics and Environment of Asthma (EGEA) survey (28) and was constructed to favor specificity over sensitivity (a job was classified as exposed only if the probability of exposure was expected to be high). For each ISCO88 code, the job exposure matrix classifies the job as exposed or not to three nonasthmagenic agents (chemicals (low level); irritants (not high peaks); exhaust fumes) and to 18 asthmagenic agents as previously described (14), further grouped as high or low molecular weight asthmagens or as mixed environments (figure 1). The asthma job exposure matrix also identifies job codes for which its exposure estimates are likely to be poorly defined. Therefore, analyses using this job exposure matrix were repeated after exclusion of jobs with such poorly defined exposures.

Data analysis

Analyses were performed on 14,151 subjects (7,255 men, 6,896 women) without missing data for asthma, job code, job exposure matrix estimates, and self-reported exposure to dusts, gases, and fumes. Analyses were performed for specific asthmagenic agents when at least 100 subjects were classified as exposed. Subjects with missing data for age at first and last asthma attacks (n = 227) were included in the analyses of “ever asthma” and “asthma with airflow limitation” but excluded in analyses of adult-onset asthma. Of these 227 subjects, 89 percent reported only a positive response to “attacks of breathlessness with wheezing” but...
not to “asthma attacks.” Compared with asthmatic subjects with age at onset after 14 years, subjects with missing values were more likely to be smokers (46 percent vs. 36 percent), less likely to have airflow obstruction (22 percent vs. 26 percent), but similar with respect to age, gender, and occupational exposure.

Attributable risks were calculated for the four definitions of asthma, considering exposure to dusts, gases, and fumes and to asthmagenic agents, and by gender, as follows: “proportion exposed × (odds ratio – 1)” divided by “(proportion exposed × (odds ratio – 1)) + 1,” using exposure prevalence estimates specific to this French population (which excludes households headed by manual workers).

RESULTS

Description of the population

Subjects were, on average, about 42 years of age (table 1). The prevalence of “ever asthma” was 7 percent in men and women and less than 2 percent for “asthma with airflow limitation.” The prevalence of adult-onset asthma was higher in women than men. A total of 25 percent of men and 20 percent of women declared themselves exposed to dusts, gases, and fumes. By design, the prevalence of exposure was similar according to the population-specific job exposure matrix. According to the asthma job exposure matrix, the prevalence of exposure to asthmagenic agents was about three times higher among women than men, whereas the prevalence of exposure to nonasthmagenic agents was about three times higher in men than women. Almost half of the jobs held by women were considered to have imprecise estimates of exposure by the job exposure matrix, with the proportion considerably less in men.

Association between job title and different definitions of asthma

A significant excess of risk of “ever asthma” was observed for stock clerks, and an excess risk of borderline significance was observed for personal care workers and restaurant...
workers (table 2), with similar results for the three other definitions of asthma (not shown). For personal care workers, odds ratios were elevated (approximately 2.5) for all other definitions of asthma, and associations were significant. Associations were significant or of borderline significance with odds ratios of $\geq 2$ for cleaners, hairdressers, laboratory aides, bakers, textile workers, leather workers, restaurant workers, stock clerks, and child care workers for at least one of the three other definitions of asthma.

**Association between exposure to dusts, gases, and fumes and asthma**

Associations between all four definitions of asthma and exposure to dusts, gases, and fumes were studied (table 3). As previously described, significant associations were found between asthma and self-reported exposure to dusts, gases, and fumes (18) but not for exposure using the population-specific job exposure matrix for dusts, gases, and fumes. Using the three other definitions of asthma, conclusions were similar, although odds ratios were slightly higher.

**Associations between asthma and specific agents using the asthma-specific job exposure matrix**

For nonasthmagenic agents identified by the asthma job exposure matrix, no associations were found (table 3), whatever the definition of asthma used (not shown). Overall, for asthmagenic agents, no significant associations with “ever asthma” were found, except for a borderline significant association for high molecular weight agents. For asthma with airflow limitation, odds ratios were higher than for “ever
asthma” for exposure to any of the following four categories (any asthmagen, high molecular weight agents, low molecular weight agents, and mixed agents), and associations were significant for all agents except high molecular weight agents. Using the two definitions of adult-onset asthma, we observed intermediate odds ratios between asthma and airflow limitation for exposure to any asthmagen.

After exclusion of subjects having jobs with imprecise estimates of exposure, all odds ratios were higher for all asthma definitions, with significantly elevated odds ratios for asthma after onset of the current job associated with any asthmagen and associated with either a high or low molecular weight agent. For high molecular weight agents, the increase in odds ratios across all definitions of asthma was greater than for the three other exposure classes. Analyses stratified by gender (not shown) indicated that the significant associations between exposure to asthmagens (all classes) identified by the job exposure matrix and asthma with airflow limitation were more pronounced among women, while associations between exposures and asthma with onset after beginning of the current job were more pronounced among men.

Association between job exposure matrix for specific asthmagen exposure estimates and asthma

Overall, results for analyses of specific asthmagens (not shown) with at least 100 subjects exposed (i.e., high molecular weight: arthropods, latex, flour; low molecular weight: highly reactive chemicals, sensitizing drugs, industrial cleaning agents, metal sensitizers; and mixed agents: textiles) were consistent with those for grouped exposures. Significant associations were found between asthma and industrial cleaning agents for all definitions of asthma and all exposure estimates, with odds ratios varying from 1.51 (95 percent confidence interval (CI): 1.05, 2.16) to 2.51 (95 percent CI: 1.33, 4.75). A significant association was observed for latex (odds ratio (OR) = 1.37, 95 percent CI: 1.02, 1.86), whereas no significant associations were found for flour and highly reactive chemicals in relation to “ever asthma.” Associations became significant between exposure to flour and highly reactive chemicals and at least two of the more specific definitions of asthma, with odds ratios about two times higher than for “ever asthma.” The associations between exposure to flour and asthma with airflow limitation and adult-onset asthma were significant only for men; of note, in this survey, all asthmatic men exposed to flour were in the lowest quintile for FEV1.

After exclusion of jobs with imprecise estimates of exposure, odds ratios increased further for latex (OR = 2.11, 95 percent CI: 1.06, 4.21) and textile production jobs (OR = 3.16, 95 percent CI: 1.13, 8.82), and associations were significant for two of the three other definitions of asthma.

Exposures to highly reactive chemicals, cleaning agents, and latex were highly correlated and could not be examined in the same model. A model including a four-class variable (not exposed to asthmagens, exposed to latex alone, exposed to highly reactive chemicals alone, exposed to cleaning agents) showed significant associations for industrial cleaning agents (OR = 1.50, 95 percent CI: 1.04, 2.18), with similar odds ratios in men and women and no associations for exposure to latex alone (OR = 1.28, 95 percent CI: 0.89, 1.85) or to highly reactive chemicals alone (OR = 0.92, 95 percent CI: 0.58, 1.48). Similar associations were found after exclusion of poorly defined jobs or using either definition of adult-onset asthma. Significant associations were found for exposure to highly reactive chemicals (OR = 2.25, 95 percent CI: 1.17, 4.33) and cleaning agents (OR = 2.16, 95 percent CI: 1.11, 1.99).
Asthma with airflow limitation

To examine the possibility that the associations seen between exposure and asthma with airflow limitation were more an indication of links between exposure and airflow limitation (than with asthma), we repeated the analyses among nonasthmatic subjects, comparing those with and without airflow limitation. No association was found between self-reported exposure to dusts, gases, and fumes and airflow limitation. Associations of borderline significance were found for exposure to dusts, gases, and fumes estimated by the population-specific job exposure matrix and for any asthmagens estimated by the asthma job exposure matrix with an odds ratio of 1.15. No associations were observed for any of the low or high molecular weight asthmagens, except for arthropods or mites, risks not found for asthma. For mixed environments, an association of borderline significance was observed with an odds ratio lower than for asthma. With two other definitions of airflow limitation (FEV1 of <80 percent of predicted value and FEV1/forced vital capacity of <75 percent), odds ratios were similar.

Attributable risk

Estimations of attributable risk for occupational exposure and asthma varied, depending on the definitions of exposure and asthma used. Considering self-reported exposure to dusts, gases, and fumes, attributable risks varied from 9 percent (“ever asthma”) to 14 percent (asthma onset after current job). Considering exposure to any asthmagens, attributable risks varied from 1 percent (“ever asthma”) to 3 percent (asthma onset after current job) and 8 percent (asthma with airflow limitation). Attributable risks also tended to increase when jobs with imprecise exposure estimates were excluded (e.g., for asthma onset after current job, attributable risk increased from 3 percent to 7 percent).

<table>
<thead>
<tr>
<th>TABLE 3. Odds ratios according to different definitions of asthma and occupational exposure, PAARC* Survey, 1975</th>
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<tbody>
<tr>
<td>Exposure to dusts, gases, and fumes</td>
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<tr>
<td>Self-reported exposure matrix</td>
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<tr>
<td>Odds ratio‡ 95% confidence interval</td>
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<tr>
<td>Exposed Nonexposed</td>
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<tr>
<td>Total Ever asthma (n = 14,151)†</td>
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<tr>
<td>Asthma with airflow limitation (n = 13,404)†</td>
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<tr>
<td>Asthma onset at/after age 14 years (n = 13,654)†</td>
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<tr>
<td>Asthma onset at/after beginning of current job (n = 13,445)†</td>
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<tr>
<td>Exposure to dusts, gases, and fumes</td>
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<tr>
<td>Self-reported</td>
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<tr>
<td>3,134 11,017 1.45 1.25, 1.67 1.51 1.17, 2.01 1.47 1.20, 1.80 1.75 1.35, 2.28</td>
</tr>
<tr>
<td>Population-specific job exposure matrix</td>
</tr>
<tr>
<td>2,952 11,199 1.02 0.87, 1.20 1.26 0.93, 1.70 1.12 0.90, 1.40 1.17 0.88, 1.56</td>
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<tr>
<td>Asthma job exposure matrix, all subjects</td>
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<tr>
<td>Asthmatics, any</td>
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<tr>
<td>2,079 12,072 1.10 0.92, 1.32 1.60 1.14, 2.23 1.15 0.90, 1.48 1.24 0.90, 1.71</td>
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<tr>
<td>High molecular weight agents</td>
</tr>
<tr>
<td>950 12,072 1.24 0.97, 1.59 1.52 0.93, 2.48 1.32 0.95, 1.85 1.21 0.77, 1.91</td>
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<tr>
<td>Low molecular weight agents</td>
</tr>
<tr>
<td>1,001 12,072 1.13 0.88, 1.44 1.61 1.04, 2.49 1.25 0.90, 1.73 1.52 1.02, 2.25</td>
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<tr>
<td>Mixed environments or agents</td>
</tr>
<tr>
<td>447 12,072 0.98 0.67, 1.43 1.89 1.03, 3.45 1.12 0.69, 1.83 1.16 0.63, 2.16</td>
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<tr>
<td>Asthma job exposure matrix, subjects with job with imprecise estimates of exposure excluded (n = 10,560)</td>
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<tr>
<td>Asthmatics, any</td>
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<tr>
<td>915 9,645 1.17 0.90, 1.51 1.98 1.28, 3.07 1.38 0.99, 1.94 1.74 1.17, 2.60</td>
</tr>
<tr>
<td>High molecular weight agents</td>
</tr>
<tr>
<td>238 9,645 1.59 1.04, 2.45 1.67 0.67, 4.14 2.03 1.18, 3.48 2.09 1.05, 4.17</td>
</tr>
<tr>
<td>Low molecular weight agents</td>
</tr>
<tr>
<td>820 9,645 1.20 0.91, 1.56 1.87 1.17, 2.99 1.40 0.98, 1.99 1.76 1.16, 2.68</td>
</tr>
<tr>
<td>Mixed environments or agents</td>
</tr>
<tr>
<td>129 9,645 1.05 0.53, 2.08 3.21 1.38, 7.46 1.39 0.61, 3.20 1.97 0.79, 4.91</td>
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</tbody>
</table>

*PAARC, Pollution Atmosphérique et Affections Respiratoires Chroniques.
† Asthmatic/nonasthmatic: ever asthma (n = 976/13,175); asthma with airflow limitation (n = 229/13,175); asthma onset at/after age 14 years (n = 479/13,175); asthma onset at/after beginning of current job (n = 270/13,175).
‡ Odds ratios were adjusted for age, smoking habits, and sex.
DISCUSSION

In summary, these analyses showed that the strength of the associations observed between exposure and asthma differed according to different definitions of exposure and asthma and that increasing the specificity of the asthma definition and the occupational exposure estimates increased the strength of associations seen. Furthermore, the results indicated a deleterious role for occupational asthmagen exposure for both low and high molecular weight agents.

Specificity and appropriate exposure estimates

These analyses showed increases in odds ratios by increasing specificity of both exposure and disease. This is consistent with other results (12–14, 25, 29). For example, in a Spanish population-based survey (25) and a Dutch survey on farmers (29), odds ratios increased after restricting the definition of asthma to include only subjects with bronchial hyperresponsiveness.

Our results suggest that it is important not only to favor specificity in the asthma definition but also to take into account relevant exposure periods for occupational asthma (14). Ideally, to estimate the association between occupational exposures and asthma, complete occupational and school histories and complete asthma history (with onset and periods of remission) are needed to identify appropriate exposures windows. This is consistent with the results of de Marco et al. (30), who studied the association between disease and exposure before and after taking into account exposure at the time of the disease (25) or subjects without changes in employers (12) and who found increased odds ratios in both cases. This is especially important for work-related asthma, as the relevant exposure is more likely the exposure in the job held at the time of asthma onset or exacerbation. However, our results indicate that, in large population-based surveys where insufficient data are available to accurately define exposure windows, taking into consideration the age at onset of asthma even in relation to the current job may provide a more valid risk estimate of the work relatedness of asthma than simply assessing “ever asthma” in relation to exposures. This was seen also in the Spanish component of the European Community Respiratory Health Survey (ECRHS), in which a higher risk was seen for occupational exposure for adult-onset asthma (25).

A limitation of this study was that it was necessary to apply the asthma job exposure matrix without the recommended step of reviewing exposure estimates for some jobs using the job title text (14). This step was designed so that known between-country differences in exposure risks could be incorporated into the estimates and to improve the specificity of estimates for very broadly defined job codes. In the initial report in which the asthma job exposure matrix was applied in the EGEA survey, analyses on about 450 subjects showed that the strength of association of asthmagen exposures with asthma increased when adding the review step (14). However, because only job codes had been retained in the PAARC Survey data set, this step was not possible here. Our results suggest that use of the asthma job exposure matrix was still valuable, even though the questionnaire contained only very basic information about job titles and only codes were available. Previous analyses of French, Dutch, and Norwegian community studies had suggested that, to apply the job exposure matrix alone (without the review step), more than 2,000 subjects are needed (20); similarly, in the ECRHS (15) with about 1,500 subjects, the odds ratios did not change appreciably after the review step. Similar to results seen in the EGEA survey in which the asthma job exposure matrix was first used (14), the odds ratios in the current analysis increased when jobs with imprecise estimates of exposure were excluded.

Self-reported exposure to dusts, gases, and fumes

The positive association observed in this study between asthma and self-reported exposure to dusts, gases, and fumes is consistent with results from other studies in which odds ratios for this association were between 1.2 and 3.0 (9, 11, 13, 18). In contrast, in a previous analysis of the PAARC Survey (18) and in the ECRHS (15), no association was found between asthma and exposure to dusts, gases, and fumes estimated by an independent job exposure matrix. This is consistent with the present negative results regarding exposure to dusts, gases, and fumes using the population-specific job exposure matrix and exposure to nonasthmagens from the asthma job exposure matrix. An association between asthma and exposure to any dusts, gases, and fumes is not a priori expected, unless there is a high prevalence of exposure to asthmagen agents in the studied population, or if there is reporting bias by asthmatic subjects. In the analysis here and in other surveys (9, 11, 18), odds ratios for the association between asthma and self-reported exposure to dusts, gases, and fumes were higher for women than for men, which is consistent with the present observation of greater exposure of women to asthmagens. In summary, these findings suggest that assessing asthma risk using self-reported exposure determined by a single imprecise question is not sufficiently accurate to be used as a sole exposure estimate, as previously reported (7, 31).

Risk of asthma according to job titles or specific asthmagens

Regarding job titles, associations found in the PAARC Survey are consistent with recent results of the reported incidence of occupational asthma in specified jobs in France (32). For cleaning workers (8, 13, 25), restaurant workers (24, 33), personnel care workers (33), and bakers (1–3), our results are consistent with those of other surveys.

Regarding the study of asthmagens, for high molecular weight agents, our results are consistent with those of the ECRHS (15), using the same asthma job exposure matrix (14), except for latex where no association was found in the ECRHS (15) and the EGEA survey (14). In 1975, subjects of the PAARC Survey were unlikely to have been exposed to latex; therefore, the associations seen here most likely reflect the effect of an associated exposure in the same jobs, such as industrial cleaning agents. For low molecular weight agents, our results are consistent with those of the EGEA survey (14), where a risk of asthma was found for subjects exposed to industrial cleaning agents and highly reactive chemicals.
For mixed environments, associations found for textile production jobs are consistent with those of other surveys (8, 12, 13, 34).

Our results and those of others suggest a deleterious role of cleaning agents on asthma (8, 13, 14, 25, 32, 33, 35) and a high risk of asthma for cleaners (10) in both private homes (35) and offices (8, 13). The use of industrial cleaning agents in 1975 among PAARC Survey subjects was also found to have a deleterious effect on both asthma and lung function (21). These associations are probably not due to the same agents in 1975 and now. Other workers, such as personnel care workers, could also be exposed to cleaning agents that are probably different from those used by cleaners. Further studies are needed to better estimate respiratory health in cleaning workers.

It is of interest that women were more exposed than men to asthmagenic agents and that the associations of asthmagenic agents with severe asthma were stronger in women than in men. Although the design of the PAARC Survey may explain part of the higher prevalence of exposure in women, it is well known that many occupations at risk, such as health-care workers, textile workers, and cleaners, are mainly female occupations (34).

In this study, asthma with airflow limitation was used as a surrogate for severe asthma, as it was not possible to study other dimensions of severity given the data available. The association between specific asthmagens and asthma severity has never been studied, and the results here suggest that further analyses are needed to examine these associations. One can hypothesize that, if some asthmagens play a role in both exacerbation and development of asthma, associations will be seen between exposures and both asthma and adult-onset asthma. In our analyses, for industrial cleaning agents, associations were significant for all asthma definitions, supporting the hypotheses that industrial cleaning agents may both induce new asthma and worsen existing asthma (34).

Finally, our finding that attributable risk estimates were lower using asthmagen exposure estimates than using exposure to dusts, gases, and fumes is consistent with results from a recent report (5) in which risks varied from 5 percent to 13 percent for occupation or exposure known to cause occupational asthma and from 14 percent to 36 percent for more general exposure to dusts, gases, and fumes. Attributable risk can be calculated only on the hypothesis of causal association, which is probably not the case for dusts, gases, and fumes in general. Our results also emphasize the variability in attributable risk estimates and their dependence on the definitions of exposure and outcome used. The attributable risk estimates in this analysis of PAARC Survey data are likely to be an underestimate of the true attributable risks, especially in men, as the population excluded manual worker heads of households.

In conclusion, our results suggest that assessment of associations between occupational exposures and asthma is improved with more precise estimates of relevant exposures and with clearer definitions of disease onset. Complete occupational and asthma histories, taking into account exposure windows according to asthma onset, are recommended. Our results also indicate that, in large population-based studies, useful information about the work relatedness of asthma can be obtained using coded job titles and an asthma job exposure matrix. An advantage of the use of job titles alone is that it allows the generation of hypotheses regarding unknown risks. Finally, to estimate attributable risk of occupational factors in asthma, accurate population-specific estimates of exposure to asthmagenic agents are needed.

REFERENCES