

Short communication

Allergic respiratory diseases and environmental pollution: experience in the printing/paper-manufacturing industry

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Several studies have shown a correlation between airborne pollutants and respiratory disorders. To determine whether professional exposure to industrial pollution might represent a risk factor for allergic respiratory diseases, we administered allergologic tests to 275 workers employed in a paper-making/printing factory and to a control population composed of 160 office workers from the same urban area. All subjects were evaluated on the basis of personal and family histories, the results of prick tests with common airborne allergens, specific serum IgE levels, pulmonary function test, and standard chest radiography. The percentage of subjects with allergies in the factory-worker group (67/275; 24.4%) was significantly higher than that observed among the office workers (20/160; 12.5%) (chi-square test: 8.17; $P < 0.01$). Of the 67 factory workers with allergies, 94% had histories of daily exposure to aliphatic hydrocarbons. The results of this study indicate that exposure to the latter type of industrial pollutants is associated with a significantly higher prevalence of allergic respiratory diseases.

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Allergic respiratory disorders are becoming more and more common, particularly in industrialized countries. Epidemiologic studies have shown that the frequency of bronchial asthma and rhinitis has significantly increased over the last 50 years, especially among young city-dwellers (1–6). The increased frequency and severity of these diseases are largely due to the environmental pollution of large cities by automobile exhaust fumes, home heating systems, and industrial emissions (7). The most common urban air pollutants are sulfur oxides (particularly SO_2), nitrogen oxides (NO and NO_2), ozone (O_3), suspended solid or liquid particles containing heavy metals, aromatic polycyclic hydrocarbons and carbon compounds (such as CO), and volatile organic compounds (primarily hydrocarbons) (8). These substances, in the form of gas or particles (noncombusted diesel fuel, tobacco smoke) produce a direct irritative effect on the airway mucosa. Numerous studies have demon-

strated a correlation between the presence of the substances in the air and the appearance of symptoms of irritation and bronchial hyperreactivity (7, 9–13). Wade & Newman recently described three cases of persistent bronchial asthma among railroad engineers exposed to massive quantities of diesel exhaust fumes (14).

Other experiences suggest that atmospheric pollutants can also promote the development of allergic respiratory diseases (rhinitis and/or bronchial asthma) by increasing sensitivity to various airborne allergens. A recent study in Japan revealed a direct relation between allergy to cedar pollen (*Cryptomeria japonica*) and traffic pollution. In areas where these trees are cultivated, the prevalence of this type of allergy was 13.2% among residents of areas that were also located near major traffic arteries, while that of persons living within cedar forests (without exposure to automobile exhaust fumes) was only 5.1%. The authors of this

report concluded that the particles contained in diesel exhaust were the main cause of the increased incidence of allergies to this pollen (15). Other investigators, using nasal and bronchial challenges with specific allergens, have demonstrated the effect of pollutants such as ozone, SO₂, and NO₂ on the development of hypersensitivity to airborne allergens (16–18).

The objective of the present study was to determine whether industrial air pollution, combined with that provoked by urban traffic, is a significant risk factor for respiratory allergies. To this end, we estimated the prevalence of hypersensitivity to a number of common airborne allergens and that of related allergic respiratory diseases in subjects who were exposed to industrial pollutants as a result of their work and in a control group of office workers from the same urban area.

Material and methods

All potential subjects were asked to complete a questionnaire on the zone of residence, the characteristics and condition of the home itself, the type of transport used to travel to and from the workplace, the amount of time spent in traffic each day, smoking habits, other professional activities associated with exposure to pollutants, and the length of time subjects had been working in their current position. On the basis of these responses, two homogeneous groups were selected that differed only in their respective degrees of exposure to industrial pollution.

The entire study population included 435 subjects, of whom 275 were workers employed in a printing and paper-manufacturing factory and the remaining 160 were office workers. All of the subjects included in the study had been working in their current positions for at least 5 years. The factory and the office building in which the control subjects worked were located in the same urban area, and there were no other significant industrial plants in this zone. The workers were recruited from various areas of the factory including the printing division (lithography, rotogravure, magnetography) and paper production and processing (wastepaper pulping, addition of minerals, bleaching, dying, paper machine, cutting). The various chemical substances (glues, metallic pigments and solvents used in inks and paints, naphtha, and aliphatic and aromatic hydrocarbons) utilized in these areas were a source of particulate pollution, and cutting operations produced a significant amount of dust. Workers on the paper production line were exposed to heat and humidity, silicate dusts (talc, kaolin), and dust originating from deposits of cellulose and wastepaper destined for pulping and stores of finished products.

During the initial screening, personal and family histories were recorded for each subject, with emphasis on symptoms and diagnosed cases of respiratory allergies. Prick tests were performed with grass pollens, absinthium, *Parietaria*, olive pollen, cat dander, *Alternaria*, *Aspergillus*, *Penicillium*, *Cladosporium*, *Candida*, *Dermatophagoides pteronyssinus*, and *D. farinae* (Dome/Hollister, Stier Diagnostics, Bayropharm Italiana, Milan, Italy), and specific IgE levels for these allergens were measured with the CAP System FEIA RAST (Pharmacia, Uppsala, Sweden). In addition, chest radiography, pulmonary function tests (Cosmed Pony Graphic Spirometer, Rome, Italy) and cytologic and bacteriologic examination of sputum were performed in all cases.

Results

Of the 275 factory workers included in the study (238 men, 37 women; mean age: 42 years), 210 worked in the graphics department, 53 worked on the paper production line (including 11 dyers), and the remaining 12 had maintenance jobs. All had been working in their current positions for 10 years or more. The mean period of employment in the factory was 20 years. The group of office workers was composed of 160 subjects (81 men, 79 women; mean age: 41 years) who had been working at their current jobs for at least 10 years and in the office building itself for a mean of 15 years. Medical histories revealed current respiratory-tract symptoms in 87 (31.6%) of the 275 factory workers (rhinitis in 66, asthma in six, and both rhinitis and asthma in 15). These problems were reported by only 39 (24.4%) of the office workers (rhinitis in 33, asthma in one, and both rhinitis and asthma in five) (chi-square test: 2.25; $P=0.1$). Seven of the factory workers (2.5%) and five office workers (3.1%) were already suffering from respiratory symptoms (rhinitis and/or asthma) when they were hired. Analysis of the family histories showed that 26 of the factory workers (9.4%) and 38 (23.8%) of the office workers had children with allergic respiratory diseases.

The allergologic examination (prick tests) revealed 180 cases of positivity among 94 (34.2%) of the 275 workers and 38 positivities in 22 (13.8%) of the 160 office workers (chi-square test: 20.5; $P<0.0001$). Correlation between reported symptoms and allergologic findings was present in 67 of the workers who presented prick-test positivity (24.4% of the entire group) and in 20 of the office workers (12.5% of the group) (chi-square test: 8.17; $P<0.01$). These “allergic” subgroups included all of the subjects with respiratory symptoms (rhinitis and/or asthma) before beginning work in the factory (seven cases) or office building (five cases).

In the factory-worker group, 23 (12.8%) of the 180 skin-test positivities were for mycophytes; other positivities observed within this group included 50 for grass pollens, 40 for mites, 39 for *Parietaria*, 24 for olive pollen, seven for absinthium, and two for cat dander. Within the group of office workers, skin-test positivities included 12 for grass pollens, nine for *Parietaria*, nine for mites, five for olive pollen, one for absinthium, one for *Alternaria*, and one for cat dander.

Discussion

The analysis of these findings and other data collected during the study revealed a significantly higher percentage of allergic (i.e., symptomatic with positive allergologic findings) subjects among the factory workers (24.4%) than the office-worker group (12.5%) (chi-square test: 8.17; $P < 0.01$). Excluding those subjects whose symptoms were already present at the time of hiring, 21.8% of the factory workers and 9.4% of the office-building employees appear to have developed allergies after beginning their current jobs (chi-square test: 10.1; $P < 0.01$). The 67 allergic subjects from the factory (58 men, nine women; mean age: 40 years) included eight who worked on the paper production line (four of whom were dyers), 53 from the graphics department, and six members of the maintenance crew. Sixty-three of these subjects (94%) (53 from graphics, four dyers, and six from maintenance) were exposed to inks, paint, and dyes (all of which contained aliphatic solvents), as well as to naphtha.

The subjects included in this study were selected by questionnaires specifically designed to reveal sources of exposure to pollutants, and the two groups were closely matched as far as nonprofessional exposure was concerned. A significant role for domestic exposure can be excluded not only on the basis of the detailed description of the home environment provided by the subjects themselves, but also by the percentages of subjects whose children suffered from perennial or seasonal rhinitis/asthma and/or had been diagnosed as having respiratory allergies (9.5% of the factory workers in comparison with 27.8% of the office workers).

The higher percentage of positive skin-test reactions to mycophytes among the factory-worker group (12.8% in comparison with 2.6% of the office workers) might raise the suspicion of occupational sensitization to these allergens, which might be expected to abound in an environment with high temperatures and humidity, such as a paper-manufacturing plant. However, all 17 of the workers that were reactive to mycophytes were also positive for at least two other allergens as well, and these cases of hypersensitivity cannot

be attributed to higher exposure levels. The increased rate of atopy among these workers thus appears to be related to their daily exposure to industrial pollutants.

There are numerous mechanisms by which chemical pollutants can promote respiratory allergies. First of all, the direct damage to the airway mucosa provoked by these agents causes structural and functional alterations in the epithelial barrier and the ciliary apparatus. These injuries lead to prolonged contact between airborne allergens and the mucosa and an increased diffusion of antigens across the epithelial membrane, thus facilitating their encounter with the immune system (19). These pollutants can also act as allergen carriers within the respiratory tract. Aggregates composed of micronic particles of atmospheric pollutants and various airborne allergens (*Fel d I*, *Der p I*, *Der f I*, *Der f II*, *Aspergillus* and *Penicillium* spores, and pollen granules) have been observed by a number of investigators (20, 21). Apart from its role in the transport of these allergens (22, 23), this association causes morphologic changes in pollen granules and modifies both the release and antigenic potential of their allergenic proteins (24). Atmospheric pollutants can also exert an adjuvant effect on the production of antigen-specific IgE against various airborne allergens, an effect that has been observed by a number of groups in animal studies (25–27). On the basis of studies conducted on cultures of human bronchial epithelial cells (28–30), it appears that pollutants such as ozone or NO₂ can increase the production of inflammatory cytokines and mediators such as leukotriene C₄, interleukin-8, GM-CSF, interferon-gamma, and ICAM-1. Finally, epidemiologic and experimental studies have shown that the extract of polyaromatic hydrocarbons from diesel exhaust particles enhances human IgE production from purified B cells (31).

In this study, the prevalence of allergic respiratory disorders among subjects exposed to industrial chemicals (i.e., aliphatic hydrocarbons) was clearly higher than that among nonexposed controls. This difference was particularly significant for mycophyte sensitivity. These findings suggest that chemical contaminants present in factory work areas may promote the development of hypersensitivity (particularly to airborne allergens also present in these environments) and related allergic respiratory diseases in workers who are exposed to these pollutants.

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