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Relationships between Indoor Environments and Nasal Inflammation in Nursing Personnel

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ABSTRACT. In this study, the authors sought to address the relationships between measured indoor environmental factors and nasal patency (i.e., minimum cross-sectional area) and volume and *markers* of nasal inflammation in nasal lavage fluid. Clinical data were obtained for 115 females who worked at 36 geriatric nursing departments. The indoor climates in the nursing departments were characterized by high room temperatures (median = 23 °C), low relative air humidities (median = 24%), and high air exchange rates indicated by low carbon dioxide levels (median = 570 ppm). Evidence of microbial amplification was observed in the ventilation unit in 3 of the departments. Decreased nasal patency was observed relative to microbial amplification in the ventilation units (minimum cross-sectional area 1 = 0.80 cm^2 vs. 0.64 cm^2, $p = .003$, minimum cross-sectional area 2 = 0.80 cm^2 vs. 0.67 cm^2, $p = .02$) and in relation to elevated indoor temperature (volume 1 = 3.46 cm^3 vs. 3.22 cm^3, $p = .03$). The authors concluded that the indoor environment may have affected the nasal mucosa of nursing personnel, thus causing nasal mucosal swelling. The results support the view that fungal contamination of air-supply ducts may be a source of microbial pollution, which can affect the nasal mucosa.
<Key words: acoustic rhinometry, *Aspergillus fumigatus,* hospital workers, indoor climate, nasal lavage>

MOST EPIDEMIOLOGICAL STUDIES on the possible health effects of indoor environments have dealt with symptom reports only. Nasal symptoms have often been studied as a part of the Sick Building Syndrome (SBS), a concept established from the observations of an increased occurrence of certain symptoms in problem buildings.[1,2]

In previous studies, an increased prevalence of mucous membrane symptoms in the eyes and upper airways among employees at geriatric hospitals has been reported.[3–5] Questionnaire studies have also demonstrated a relationship between symptoms and elevated room temperature (i.e., above 22–23 °C),[6–8] airborne dust exposure,[7,9] and dampness in buildings.[10]

Recently, more objective methods for studying environmental effects on the mucous membranes of the upper airways have been developed. Acoustic rhinometry (AR) has been used for the measurement of nasal patency.[11] Investigators have used nasal lavage (NAL) to measure several biomarkers of inflammation in the nasal mucosa, such as tryptase, albumin, lysozyme, eosinophilic cationic protein (ECP), and myeloperoxidase (MPO). Increased nasal mucosal swelling, observed with AR, has recently been associated with low air-exchange rates[12] and exposure to allergens,[13] dust,[14] environmental tobacco smoke,[15] and volatile organic compounds.[16] A relationship between poor cleaning routines,[17] low air-exchange rates,[12,17] mechanical ventilation,[12] and increased concentrations of ECP and lysozyme in NAL has also been demonstrated. However, only low correlations have been found between nasal symptoms and such measurable signs of nasal inflammation.[14,18]

With respect to the indoor environment of buildings, there have been extensive investigations, and increased attention has been paid to particulate pollution and cleaning practices.[19,20] Methods for the assessment of dust-settlement rates and cleanliness have been developed; dust is collected on foil, on which the amount of dust is determined as the area covered by dust (i.e., percentage of total surface area). A guideline for indoor environment quality proposed 1–1.5% as a limit for percentage of total surface area.[21] In buildings with mold growth in the ventilation system or on internal surfaces, increased counts of fungi may be observed, and there is a concomitant increase in species of *Aspergillus* and *Penicillum*[22] in particular.

In this study, we investigated whether there was a relationship between indoor environmental factors and selected measures of nasal patency and inflammation.

Material and Method

Subjects. This investigation was part of a study of complaints about the indoor climate among staff at geriatric hospitals in the municipality of Trondheim (145,000 inhabitants), which is located in the middle of Norway (i.e., 63 °N). A self-administered questionnaire, which contained symptom questions identical to those of the MM-040B Örebro-questionnaire,[23,24] was used for the identification of subjects with and without mucous membrane symptoms (MMS). The questionnaire was posted to all female employees (n = 1,165) at the nursing departments of 14 geriatric hospital units during the spring of 1995. There were 821 persons (70%) who responded.

Among the respondents, 80 females had reported nasal symptoms every week during the preceding 3 mo, whereas 144 had reported no such symptoms, thus giving a total of 224 subjects. Of the 224 subjects, 116 (52%, 52 with and 64 without nasal symptoms) volunteered to participate in clinical examinations. One subject was excluded from the study because of fever during the week prior to the examination.

Clinical examinations of the participants were performed at the Department of Occupational Medicine at the University Hospital of Trondheim, from November 1995 to March 1996, a period characterized by cold weather (monthly mean temperature = –3 to +1 °C), thus necessitating artificial heating. The temperature at the Department of Occupational Medicine was constant during the clinical examinations (22.3 ± 0.3 °C), but the indoor relative humidity varied, mainly depending on the outdoor temperature (range = 12–35%).

Personal characteristics. The median age of the participants was 42 yr (range = 22–64 yr), and 40 (35%) participants were current smokers. There were 17 (13%) subjects who, in the initial questionnaire, stated that they had had a history of childhood eczema. Among the nonparticipants, 8 (7%) reportedly had a history of childhood eczema and 62 (57%) were current smokers. The median age of the nonparticipants was 42 yr (range = 20–64 yr).

Information about current nasal symptoms (i.e., runny nose, sneezing, and nasal obstruction) during the week prior to the examination was gathered at the time of examination. A total of 60% of the participants reported having 1 or more such symptoms during the week prior to the clinical examination. The most common symptom was nasal obstruction (46%). Runny nose and sneezing were reported in 37% and 30% of the participants, respectively.

The age distribution, incidence of childhood eczema, and indoor smoking differed between the nursing departments and, therefore, might have been potential confounding factors.

Assessments of biological outcome. Biological outcomes were assessed by the concentration of inflammatory markers in NAL and by AR. In the NAL, concentrations of ECP, albumin, IL-2, IL-4, IL-5, and IFN-γ were measured. The AR was used for the determination of nasal patency.

NAL was performed at room-temperature (i.e., 20–22 °C) with isotonic saline solution. Five milliliters of the solution were slowly (1 ml/sec) flushed into the nostril with a plastic syringe mounted on a nose adapter (Näsoliv, 18 or 22 mm, FMAB–Förbandsmaterial AB [Partille, Sweden]). The solution was installed and suctioned back into the syringe 5 times for each nostril. The lavage fluid from the 2 nostrils was then mixed and centrifuged at 600 g for 5 min. The supernatant was then centrifuged for another 10 min at 1,200 g. One milliliter of the supernatant was frozen at –135 °C.

The NAL concentrations of cytokines IL-2, IL-4, IL-5, and IFN-γ were determined by ELISA (IL-2, IL-4, and IFN-γ—BIOTRAK,™ Amersham [International Place, England], and IL-5—Pharmingen [California]).[25] All samples were analyzed in the same run. The analysis of the albumin content of the NAL fluids was based on the binding of albumin to bromocresol green with subsequent photochemical detection.[26]

In the statistical analysis of the results, the concentrations of ECP and albumin were treated as continuous variables. The detection limit of ECP and albumin measurements was 2 µg/l and 10 mg/l, respectively. The halves of the detection limits were set to samples with

concentrations below the detection limit. In the analysis, we treated the concentrations of cytokines as dichotomous variables and used the detection limits, which varied between < 1.0 ng/l and 32.5 ng/l, as cut-off points.

AR (Rhin2000, wideband noise, continously transmitted, ver. 1.27, RhinoMetrics AS [Lyngby, Denmark]) was performed on each individual with a rhinometer probe fitted with an anatomical nose adapter. The measurements were performed at least 20 min after nasal lavage. Each examination included 3 valid readings from each nostril.

In the analysis of the results from the AR, the minimum cross-sectional areas (MCA) and the volume (VOL) between 0 and 22 mm from the opening of the nostril (MCA1, VOL1) and between 22 and 54 mm (MCA2, VOL2) were registered, together with the cross-sectional areas from 0 through 80 mm. We calculated the values for the MCA and VOL by adding the average of the 3 valid readings from each side.

Environmental factors. The 14 geriatric hospitals included 18 buildings that housed 36 nursing departments. The ages of the buildings ranged from 3 to 72 yr. There was no humidification or air-cooling device in any of the buildings. Indoor smoking was partly restricted. In some departments smoking was restricted to certain areas; in other areas, residents were allowed to smoke in the main living areas and/or in their rooms. In general, staff were prohibited from smoking indoors. Some visible signs of dampness or microbial growth were observed in 2 of the buildings. The size of the main living areas in the departments varied between 125 m^2 and 635 m^2 (median = 351 m^2).

The measurable indoor factors considered in the study were temperature, relative humidity, carbon dioxide, dust settlement rate, and microbial flora. All measurements were performed in the same period as were the clinical examinations.

Outside the hospitals, outdoor temperature and relative humidity (Vaisala Humidity & Temperature indicator HMI 31 Vaisala OY [Helsinki, Finland]) were measured. Inside in the main living area, temperature, relative humidity, and carbon dioxide (Telaire koldioxidemätare, Telaire Europe AB [Delsbo, Sweden]) were measured. The measurements were made in each department at 3 different locations (i.e., 1.1 m above the floor) over periods of 4–6 hr.

Measurements of the dust settlement rate were made on standardized steel plates (1–1.5 m above the floor), which collected dust for 1 wk. The measurements were performed at 2 different locations in each department. The average of 3 samples from each steel plate was used. The surface dust was measured by a laser extinction meter (BM-Dustdetector, N.P. Kloch [Nivå, Denmark]) and was expressed as the area covered by dust in the percentage of the total surface area.

Viable microbiological samples were collected with a centrifugal air sampler (Biotest RCS sampler, Biotest [Solihull, United Kingdom]) and agar strips (Hycon Agar Strips, Biotest [Solihull, United Kingdom]). The substrates used for sampling were Tryptic Soy Agar (total counts) and Rose-Bengal-agar for molds. The effective sampling volume was 40 l/min for particles with an aerodynamic diameter equal to 4 µm. The sampling time was 8 min per agar. At least 2 sampling locations in each department were chosen; 1 in the middle of the living room and 1 close to the exhaust air duct. If the room had mechanical air supply, a 3rd air sample was taken in the air inlet. As a reference, an air sample taken outside the building was used. Viable molds and bacteria were determined by incubation at room temperature (22 °C) and at 37 °C for 1–2 wk. We estimated the detection limit for viable organisms as 100 colony forming units (cfu) per m^3 of air.

Indoor samples, which did not resemble the microbial flora present on the outdoor sample taken at the same location, were noticed. A dominance of 1 or 2 species was regarded as a sign of microbial amplification in the building or ventilation unit.

In addition to measurements, hospital vicinity was included as a proxy variable for traffic pollution. We considered vicinity to heavy trafficked roads as a proxy for traffic pollutants in the indoor environment. Four of the hospitals were situated in the inner city, and a 5th was located near a main road outside the city hub, where traffic was the main source of outdoor air pollution. The hospitals were located near roads that had an approximate traffic density of 20–25,000 vehicles per 24 hr.

Statistical analysis. The relationships between environmental factors and clinical findings were initially addressed with bivariate analyses. Kendall's rank correlation tests or the Mann-Whitney *U* tests were used, when appropriate.

We applied multiple linear-regression analyses to address possible relationships between the environmental and biological measures, with adjustment for possible confounding factors such as age, childhood eczema, and current smoking. In the multiple-regression models, we logarithmically transformed albumin and ECP concentrations to obtain a closer approximation to normal distribution of the residuals. In additional analyses, adjustment was also made for the selection procedure (e.g., nasal symptoms) reported in the self-administered questionnaire. Collinearity diagnostics were applied for the multiple-regression models.[27] A high correlation between coefficients in the models and a tolerance below .2 were used as indicators of

Table 1.—Median Values and Interquartile Ranges of Temperature, Levels of Relative Humidity, Carbon Dioxide, and Dust Settlement Rate in 36 Nursing Departments of Geriatric Hospitals in the Municipality of Trondheim

Environmental factors	Median	Interquartile range
Temperature (°C)	23	23–24
Relative humidity (%)	24	17–26
Carbon dioxide (ppm)	570	490–650
Dust settlement (% surface/wk)	1.6	1.3–2.2

collinearity problems. A 5% level of significance was used in all statistical analyses. All the data were analyzed by the Statistical Product and Service Solutions (SPSS) for Windows, release 9.0.1 (1998; SPSS Inc., Chicago, Illinois).

The protocol of the study was approved by the Ethical Committee for Medical Research in Mid-Norway and the Norwegian Data Inspectorate Board.

Results

Environmental factors. The climatic conditions at the nursing departments at the time of clinical examinations were characterized by high indoor temperatures, which ranged from 22 °C to 25 °C. The outdoor temperature ranged from –12 °C to +5 °C (median = 0 °C). Median values for indoor temperatures, relative humidities, levels of carbon dioxide, and dust settlement rates are given in Table 1. In most of the departments, the indoor microbial flora resembled the flora of the outdoor samples, which typically constituted a mixture of bacteria, molds, and yeast, such as *staphylococci, micrococci, Cladosporium, Penicillum,* and *Aspergillus.* In samples from 2 air inlets, *A. fumigatus* was the only species identified. We, therefore, assumed that this was an indication of a possible microbial amplification of *A. fumigatus* in the ventilation unit. One of these units supplied air to 2 nursing departments. There were no indications of abnormal indoor microbial flora or *A. fumigatus* in the 2 buildings, in which some visible signs of dampness or microbial growth were evident in the building construction.

Table 2.—Median Values and Interquartile Range Recorded for Rhinometric Measurements and Albumin and ECP in Nasal Lavage Fluids in 115 Female Personnel at Geriatric Hospitals in the Municipality of Trondheim

Biological outcomes	No. of valid measurements	Median	Interquartile range
MCA1 (cm^2)	110	0.79	0.66–0.91
VOL1 (cm^3)	110	0.78	0.67–0.93
MCA2 (cm^2)	110	3.27	2.96–3.64
VOL2 (cm^3)	110	6.09	5.03–7.10
Albumin (mg/l)	100	26	18–39
ECP (mg/l)	114	< 2	< 2–3

Notes: ECP = eosinophilic cationic protein, MCA = minimum cross-sectional area, and VOL = volume.

Biological outcomes. Biological findings from the clinical examination regarding nasal patency and the concentration of albumin and ECP in NAL fluid are summarized in Table 2. IL-4 and/or IL-5 were detectable in 15 subjects, and IFN-γ was detectable in 13 subjects. Among these subjects, all 3 cytokines were detected in 6 of these subjects. Levels of IL-2 were not above the detection limit.

Relationships between environmental and biological measures. Possible relationships between environmental and biological outcomes were initially addressed with bivariate analyses. Median values for biological outcomes across 3 environmental factors are given in

Table 3.–Median Values and Interquartile Range (IQR) Recorded for Rhinometric and Nasal Lavage Measures across 2 Categories of the Variables Microbial Flora (Presence vs. No Presence of *A. fumigatus*), Temperature (< 23.2 °C and > 23.2 °C), and Hospital Vicinity (With or Without Heavy Traffic) in the Study Population of 115 Female Personnel at Geriatric Hospitals in the Municipality of Trondheim

Outcome variables	*A. fumigatus*			Temperature			Heavy traffic vicinity		
	Not present (n = 92)	Present (n = 18)	p value*	< 23 °C (n = 53)	> 23 °C (n = 57)	p value*	No (n = 71)	Yes (n = 39)	p value*
MCA1 (cm^2)									
Median	0.80	0.64	.003	0.81	0.79	.006	0.80	0.77	.81
IQR	0.69–0.93	0.47–0.79		0.69–0.93	0.64–0.90		0.68–0.91	0.59–0.97	
MCA2 (cm^2)									
Median	0.80	0.67	.02	0.82	0.76	.13	0.78	0.70	.46
IQR	0.68–0.98	0.49–0.85		0.68–1.00	0.66–0.93		0.68–0.90	0.62–0.90	
VOL1 (cm^3)									
Median	3.34	3.19	.08	3.46	3.22	.03	3.32	3.24	.64
IQR	2.97–3.71	2.94–3.29		3.13–3.70	2.94–3.64		2.97–3.68	2.90–3.59	
VOL2 (cm^3)									
Median	6.12	6.08	.29	6.29	6.08	.30	5.87	6.26	.31
IQR	5.09–7.32	4.75–6.63		5.23–7.31	4.89–6.73		5.03–6.95	4.90–7.75	
Albumin (mg/l)									
Median	27	23	.28	30	22	.007	29	22	.006
IQR	18–39	15–36		23–44	17–31		21–43	15–27	
ECP (µg/l)									
Median	< 2	< 2	.55	< 2	< 2	.80	< 2	< 2	.28
IQR	< 2–3	< 2–8		< 2–3	< 2–3		< 2–4	< 2–3	

Notes: MCA = minimum cross-sectional area, VOL = volume, and ECP = eosinophilic cationic protein.
*Mann-Whitney *U*-test, 2-tailed.

Table 4.—Two Multiple Linear Regression Models with Partial Regression Coefficients (*b*) and 95% Confidence Intervals (CIs) for the Relationships between the 2 Determinants; Presence of *A. fumigatus* in Air from Air-supply Units (Present vs. Not Present) and Temperature (°C) and Rhinometric Outcomes

Outcome variables	*A. fumigatus*			Temperature		
	b	95% CI	*p* value	*b*	95% CI	*p* value
MCA1 (mm^2)	−15	−26, −4	.007	−6	−10, −1	.02
MCA2 (mm^2)	−25	−25, −1	.03	−6	−11, −1	.03
VOL1 (mm^3)	−199	−439, −41	.10	−86	−191, −19	.11
VOL2 (mm^3)	−657	−1,560, −246	.15	−426	−816, −36	.03

Notes: In both models, 3 potential confounding factors (i.e., age, childhood eczema, and current smoking) were included. The unit of the regression coefficient (b) is that of the outcome variable per unit change in the independent variable. MCA = minimum cross-sectional area, and VOL = volume.

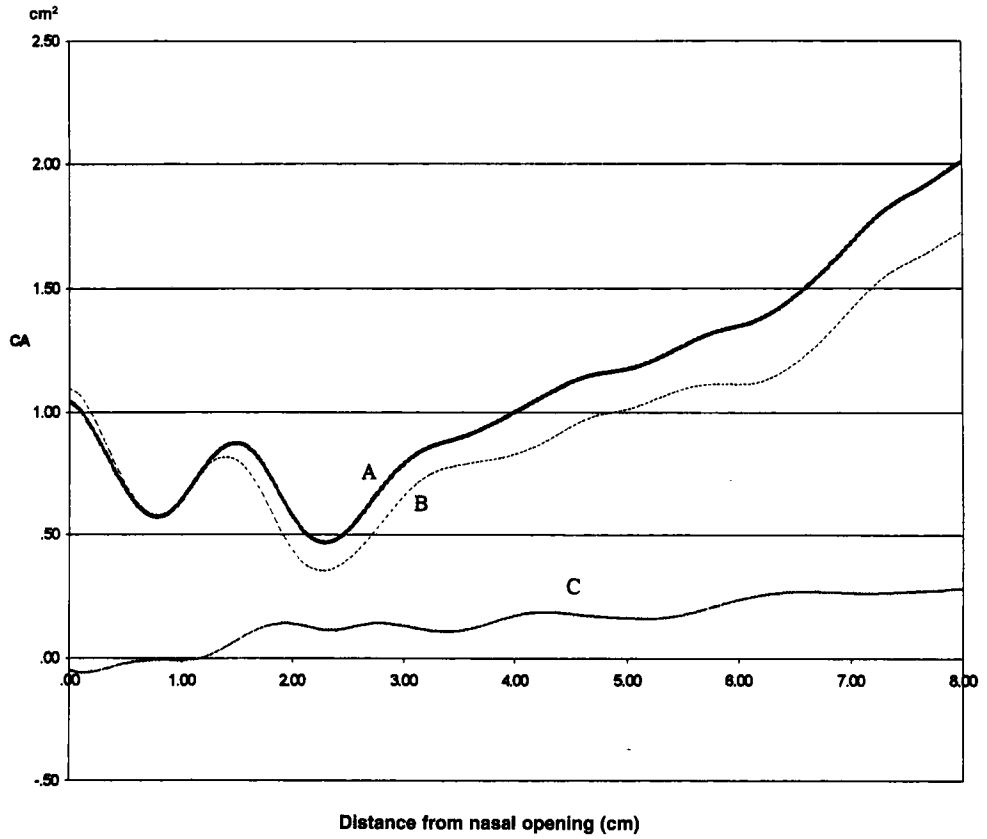

Fig. 1. Mean acoustic rhinometry curves in personnel working in hospitals without (curve A) and with (curve B) indications of microbial amplifications in the ventilation unit of *A. fumigatus*. Curve C shows the difference between curve A and curve B.

Table 3. No statistically significant differences in the biological outcomes were observed related to dust settlement, carbon dioxide, or relative humidity. For both temperature and presence of *A. fumigatus* in the ventilation supply air, negative and statistically significant relationships were observed with rhinometric measures.

The relationships between the presence of *A. fumigatus* and temperature and rhinometric measures were also addressed with multiple-regression analysis, and we controlled for possible confounding factors (i.e., age, childhood eczema, and smoking habits). The analyses show that subjects working in hospitals with *A. fumigatus* and high room temperatures had reduced cross-sectional areas and volumes, as indicated by negative partial regression coefficients (Table 4). The mean acoustic rhinometry curves in personnel working in hospital units without (curve A) and with (curve B) presence of *A. fumigatus* in air from supply units are given in Figure 1. Curve C shows the differences between curve A and curve B.

Except for the observed relationships described above, no other statistically significant relationships between measured environmental factors and nasal patency, or the markers of inflammation in the NAL fluid (i.e., IL-2, IL-4, IL-5, IFN-γ, albumin, and ECP) were found in the bivariate or multivariate statistical analyses. No collinearity problems were indicated in any of the multivariate regression analyses. When additional control for the selection procedure was performed, no significant impact was observed on the results.

Discussion

Our results lend support to the view that environmental factors may affect nasal patency and concentration of some inflammatory markers in NAL fluid. The most consistent findings were decreased nasal patency in relation to (1) presence of *A. fumigatus* in ventilation supply and indoor air and (2) elevated room temperature.

The study was performed in a sample of subjects with and without nasal symptoms. Selection bias resulting from this sampling strategy was, in our opinion, probably not too serious inasmuch as (1) clinical signs that had a low correlation with nasal symptoms were compared with objective environmental measurements and (2) additional control for the selection procedure did not show any significant impact on the results.

Some selection bias from the low participation rate (52%) might have occurred. The low participation rate might be explained in part by changes in the workforce between administration of the initial questionnaire (April-May) and the subsequent clinical examination (November-March). Reasons, such as change of job or long-term leave owing to sickness or to pregnancy, were verified in 20 subjects. In addition, some of the head nurses were unwilling to allow personnel to participate in the clinical examinations during work hours (verified in 9 subjects). Therefore, as there are few indications of a selection bias resulting from the seriousness of symptoms, we do not think this potential bias has seriously hampered the results.

In this study, we did not aim to study the relationships between nasal symptoms and personal factors (e.g., age, childhood eczema, smoking habits, physiological or biochemical markers of nasal mucosal inflammation). However, no statistically significant correlations were observed between these factors and the studied physiological markers, and adjustment for these factors did not change the observed relationships between environmental factors and decreased nasal patency. Other authors[18,28] have reported only a weak correlation between nasal symptoms and physiological or biochemical measurements.

If we compare our data with those of other recent studies from Scandinavia, our hospital staff had smaller nasal cavity dimensions and higher levels of albumin in the NAL fluid.[12,29] We also found that nasal patency decreased as room temperatures increased. These findings are similar to those reported by schools[12] (i.e., decreased nasal patency at temperatures above 22 °C). Compared with these school environments, the indoor climates at our geriatric hospitals were characterized by higher room temperatures and lower relative humidity. The low indoor relative humidity resulted from the high room temperature; the low outdoor temperature (typical for the winter climate in North Scandinavia); and high air exchange rates, as indicated by low indoor levels of carbon dioxide.

The nasal patency in subjects working at hospitals situated in areas with heavy traffic might have been influenced by traffic pollutants. During the winter, levels of traffic pollutants in these areas in Trondheim periodically exceed the national air quality guidelines of 75 $\mu g/m^3_{24\text{-hr average}}$ for NO_2, 35 $\mu g/m^3_{24\text{-hr average}}$ for PM_{10}, and 20 $\mu g/m^3_{24\text{-hr average}}$ for $PM_{2.5}$, respectively. Occasionally, concentrations above 185 $\mu g/m^3_{24\text{-hr average}}$ for NO_2, 330 $\mu g/m^3_{24\text{-hr average}}$ for PM_{10}, and 70 $\mu g/m^3_{24\text{-hr average}}$ for $PM_{2.5}$ are observed.[30] A greater effect of heavy traffic has also been observed among Swedish school employees, in which increased signs of nasal inflammation were observed among personnel at schools situated near roads with heavy traffic, compared with personnel who work at schools situated in areas without heavy traffic.[31]

The observed decrease in the nasal patency in personnel who worked in departments where there was *A. fumigatus* in the ventilation supply air might be caused by an increased inflammatory reaction in the nasal mucosa, thus causing an increased swelling of the mucosa, but we found no increase in ECP or albumin. This increased swelling might result from a specific allergic[32] and/or non-allergic[33] inflammatory reaction due to exposure to mycotoxins or β-1→3-glucanes.[22] Decreases in nasal patency were also recently reported in teachers working in buildings that had an identified presence of *Aspergillus spp.*[31] Norbäck et al.[31] also reported increased levels of inflammatory markers (e.g., ECP and lysozyme in nasal lavage fluid from teachers in the same environments).

We conclude, therefore, that the indoor environment may affect the nasal mucosa (i.e., mucosal swelling). The results support the view that fungal contamination of the air-supply ducts may be a source of microbial pollution, which can affect the nasal mucosa. The findings illustrate the significance of good building maintenance and prevention of microbial growth in ventilation systems. The results also suggest that lowering room temperatures could have a beneficial effect on the nasal mucosa.

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