Airways Inflammation and Glucan in a Rowhouse Area

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A study was undertaken in a number of rowhouses, some of which had had previous problems related to dampness and water leakage. The aim of the study was to assess the relation between exposure to airborne (1→3)-β-D-glucan, a cell-wall substance in molds, and airways inflammation. The study involved 75 houses with indoor (1→3)-β-D-glucan levels ranging from 0 to 19 ng/m^3. Of 170 invited tenants, 129 (76%) participated in the study. A questionnaire relating to symptoms was used, and measurements were made of lung function and airway responsiveness. Myeloperoxidase (MPO), eosinophilic cationic protein (ECP), and C-reactive protein (CRP) were measured in serum. Atopy was determined with the Phadiatop test. The major findings were a relation between exposure to (1→3)-β-D-glucan and an increased prevalence of atopy, a slightly increased amount of MPO, and a decrease in FEV_1 over the number of years lived in the house. The results suggests the hypothesis that exposure to (1→3)-β-D-glucan or molds indoors could be associated with signs of a non-specific inflammation. Thorn J, Rylander R. Airways inflammation and glucan in a rowhouse area.

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Numerous investigations in different countries have reported relationships between dampness/mold growth indoors and airway symptoms among adults and children (1–10). The symptoms generally consist of irritation in the eyes, nose, and throat; dry cough; headache; tiredness; and skin problems. Initially, the symptoms were referred to as "sick building syndrome" but, with greater knowledge of the pathology causing them, and through comparisons with symptoms among persons occupationally exposed to different organic dusts (11), it is becoming increasingly clear that most of them reflect a nonspecific inflammation in the airways.

Molds and bacteria, commonly present in increased numbers in damp houses, contain several substances that have inflammatory properties. The most studied of these are bacterial endotoxin and (1→3)-β-D-glucan, a polyglucose compound in the cell-walls of fungi, certain bacteria, and plants (12, 13).

There is a well-established relationship between humidifiers contaminated with gram-negative bacteria/endotoxin and inhalation fever (toxic pneumonitis) (14). Endotoxin produces a neutrophil-dominated inflammation in the airways and increased airway responsiveness. A relation between the severity of asthma and endotoxin levels in house dust has been reported (15). Guidelines for airborne endotoxin exposure in the environment have been suggested (16).

Regarding (1→3)-β-D-glucan, relationships have been demonstrated with the extent of symptoms of airway inflammation in exposed populations (17, 18). Experience in animal inhalation experiments demonstrates that (1→3)-β-D-glucan does not cause a neutrophil inflammation, but that it influences macrophage functioning and acts synergistically with other inflammatory agents, particularly bacterial endotoxin (19, 20).

The purpose of this investigation was to assess the relationship between the amount of airborne endotoxin and (1→3)-β-D-glucan in indoor environments and the presence of airways inflammation, in terms of symptoms, airway responsiveness, markers for inflammation in serum, and atopy. The study was approved by the Ethics Committee of the Faculty of Medicine in Gothenburg.

METHODS

The study was conducted in a rowhouse area in Gothenburg, Sweden. The houses were constructed in the 1960s, and there were numerous complaints in some of the houses over the intervening years about dampness originating from the ground or leaking roofs. Other houses were without problems. Symptoms related to the airways, fatigue, and odors of molds were frequently reported by some of the tenants.

Population Sample

The study base comprised all tenants who rented their houses and were registered in the tenant roster. They had to be 18 yr old or more, and had to have lived in their house for at least 1 yr. Of the 170 tenants invited, 129 (76%) consented to participate. They were investigated from February to April and in November 1996. For an individual subject, all investigations were made on the same day. A blood sample was taken, and the subject was then interviewed, using a standard questionnaire. Following this, lung function and airway responsiveness were measured.

There were 67 (52%) female and 62 (48%) male subjects included in the study. Of these, 34 (26%) were smokers and nine (7%) had physician-diagnosed asthma. The mean age of the subjects was 57 yr, with a range of 18 to 83 yr, and 50 subjects were 65 yr old or more (39%). The average number of years the subject had lived in their houses was 18 yr, with a range of 2 to 36 yr. In some houses, more than

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one test subject was recruited. Such persons were equally distributed among homes with low and high levels of (1→3)-β-D-glucan.

Spirometry and Airway Responsiveness

Spirometry was done with standard techniques. A Vitalograph model S (Vitalograph Ltd., Buckingham, UK) with a PFT printer (Matsushita, Japan) was used and calibrated every morning with a 1-L syringe. The test subjects performed at least three technically acceptable trials according to American Thoracic Society (ATS) criteria, and the largest value for FEV_1 was registered and compared with predicted values (21).

Airway responsiveness was assessed with a methacholine (MCh) challenge according to Yan and colleagues (22), with some modifications. Spirometry was performed to exclude persons with less than 70% of predicted values in FEV_1 and/or FEV_1/FVC. The subjects initially inhaled one dose of saline. The spirometric values obtained 1 min after this inhalation were used as baseline values. The MCh (3 μl/inhalation) was administered in increasing doses at 3-min intervals, to a total amount of 1.2 mg. The maximum FVC and FEV_1 were recorded 1 min after each dose of MCh. In cases in which FEV_1 decreased by more than 10% from the baseline value after one dose, dose levels were increased more slowly. If FEV_1 decreased by more than 20% from the baseline value after any dose of MCh, the test was discontinued. The results were expressed as the group average decrease in FEV_1 after the highest dose of MCh given. A decrease in FEV_1 was identified as an increase in airway responsiveness.

Questionnaire

The subjects were interviewed with a slightly modified standard questionnaire for the assessment of organic-dust-induced effects (17, 18). The questionnaire contained a series of items on existing diseases, occupation, length of time the subject had lived in the present house, and the presence of pets. Questions were also posed about odors of molds and spots of dampness in the house. These were followed by a series of questions about different symptoms present during the most recent 3 mo. The symptoms were cough, dry or with phlegm; chest tightness; shortness of breath; irritation in the eyes, nose, or throat; and nose congestion and itchy nose. Questions were also posed on joint pains, muscle pains, headache, unusual tiredness, wheezy chest, and skin problems. Special questions dealt with subjective airway reactivity, chronic bronchitis, asthma, and episodes of fever and influenza-like symptoms that were gone the next day. The questionnaire was concluded with questions about physician-verified allergy and smoking habits. Chronic bronchitis was defined as cough with sputum for at least 3 mo a year for a period of at least 2 yr. Asthma was defined as physician-diagnosed asthma.

Inflammatory Markers and Atopy

Eosinophilic cationic protein (ECP), myeloperoxidase (MPO), and C-reactive protein (CRP) were measured in serum (Clinical Chemistry Laboratory, Sahlgren's Hospital, Gothenburg). ECP was assayed with the fluorescence enzyme immunoassay (FEIA) technique (CAP ECP FEIA; Pharmacia Diagnostics AB, Uppsala, Sweden) and expressed as μg/L. MPO was measured with a radioimmunoassay (RIA) (CAP MPO RIA; Pharmacia Diagnostics AB) and expressed as μg/L. CRP was assayed according to the Mancini technique (Behring, Frankfurt, Germany) and expressed as μg/ml.

The concentration of serum IgE antibodies against 10 airborne allergens was assayed with the FEIA technique (CAP Phadiatop FEIA, Pharmacia Diagnostics AB). The results were expressed as positive (atopic) or negative (nonatopic).

Exposure

Measurements of airborne dust for determinations of (1→3)-β-D-glucan and endotoxin were made in 75 houses. Airborne dusts were generated by using a machine designed to generate dust equivalent to that generated by a few people moving about the room (18). More than 90% of the particles generated were in the size range of 0.5 to 3 μm as measured with a laser particle counter (Met One, Inc., Grants Pass, OR). Two rooms were investigated in each house, and two filters were placed in each room.

Air samples were taken by drawing air through Isopore filters (ATTP 0.8 μm; Millipore, Cambridge, MA) at a flow rate of 5 L/min for 30 min. For analyses, the filters were shaken for 10 min in 10 ml pyrogen-free water, and a sample was set aside for later endotoxin analyses. Following this, 0.3 M NaOH was added and the filters were shaken on ice for 10 min to unwind the triple-helix structure of the glucan and make it water soluble. The extracts were analyzed for the amounts of (1→3)-β-D-glucan and endotoxin through the use of specific *Limulus* lysates (23). Filter-extract samples of 50 μl were placed in a microwell plate, and 50-μl of specific glucan lysate (Fungitic G Test; Seikagaku Co., Tokyo, Japan) or specific endotoxin lysate (Endospecy; Seikagaka Co.) was added. The plate was incubated in a spectrophotometer (Scinics Corp., Tokyo, Japan), and the kinetics of the ensuing color reaction was read photometrically, transformed into absorbance units, and compared with a standard curve. The results were expressed as ng/ml liquid. Using the value for air flow through the filter, the results were then transformed into units of ng/m^3. The detection limit for this technique is 10 pg/ml for endotoxin and 20 pg/ml for (1→3)-β-D-glucan. The CV for the method is 1.22%. The results were expressed as the mean of the values for the four filters used in the study.

Treatment of Data

The differences between effect variables of persons living in houses with different levels of (1→3)-β-D-glucan were analyzed with Student's *t* test, one-way analysis of variance (ANOVA), and nonparametric tests (chi-square, Fisher's exact test, and the Mann–Whitney U test) for comparison of group means. Logistic-regression analyses were performed to evaluate the relationships between indoor (1→3)-β-D-glucan exposure and the different effect variables. Odds ratios (ORs) with 95% confidence intervals (CIs) were computed while controlling for age, gender, cigarette-smoking status, pets, and atopy. ORs for the continuous variables were calculated as the risk for an increase of 1 SD (24). Linear regression analyses were performed to evaluate the relationship between the baseline FEV_1 and the number of years lived in the house, while controlling for age, gender, cigarette-smoking status, pets, asthma, and atopy. When the number of subjects was small, the analyses were limited to bivariate correlation tests (Pearson's and Spearman's tests) and partial correlation tests with control applied for age, asthma, atopy, cigarette-smoking status, gender, and pets. Separate analyses of atopic and nonatopic subjects were also performed. Differences were considered statistically significant at $p < 0.05$.

RESULTS

Environmental Measures

No detectable amounts of endotoxin were found when analyzing the first 20 filters, and analyses for endotoxin were therefore not performed on the remaining filters. Among these, there were filters containing low and high amounts of airborne (1→3)-β-D-glucan. The distribution of airborne (1→3)-β-D-glucan levels in the different houses is shown in Figure 1.

In the 75 houses, indoor (1→3)-β-D-glucan levels ranged from 0 to 19 ng/m^3. Of the 75, 20 houses had (1→3)-β-D-glucan levels below 1 ng/m^3 and 13 houses had levels above 6 ng/m^3.

There were no significant differences between subjects reporting mold odors as compared with those not reporting such odors with regard to the amounts of (1→3)-β-D-glucan in their bones (3.9 versus 3.4 ng/m^3, p = NS), nor between subjects reporting spots of dampness in the houses as compared with those not reporting such spots (3.3 versus 4.0 ng/m^3, p = NS).

Atopy

A total of 24 subjects (19%) had a positive Phadiatop test. There were no differences between groups when subjects were divided into those with levels higher and levels lower than 1 ng of (1→3)-β-D-glucan/m^3 (18.5%, n = 17, versus 18.9%, n = 7). When the division was made according to those

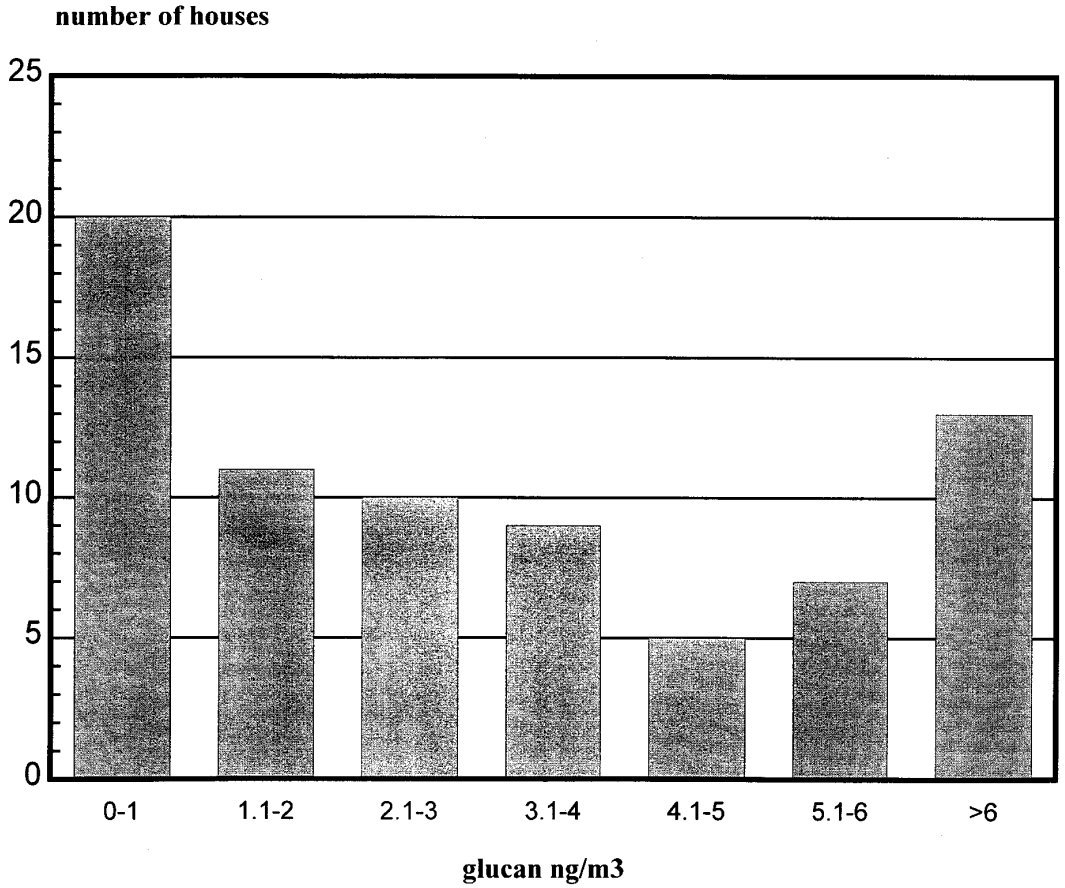

Figure 1. The distribution of airborne (1→3)-β-D-glucan in the houses investigated.

with (1→3)-β-D-glucan levels higher and lower than 3 or 4 ng/m^3, the proportion of atopic subjects was larger in the high-exposure groups, but the differences were again not statistically significant (24.6%, n = 15, versus 13.2%, n = 9; and 26.7%, n = 12, versus 14.3%, n = 12). The results were similar among nonsmokers and nonsmokers/nonasthmatic subjects (data not shown).

Analyses of all subjects under 65 yr of age showed more or less similar results (data not shown). Among subjects older than 65 yr, however, the proportion of atopic subjects was significantly larger in the high (> 3 ng/m^3)-exposure group than in the low (< 3 ng/m^3)-exposure group (32.0%, n = 8, versus 4.5%, n = 1, p = 0.02).

Spirometry and Airway Responsiveness

The baseline FEV_1 values were unrelated to amounts of (1→3)-β-D-glucan for either the whole group or for atopic or nonatopic subjects.

The decrease in FEV_1 after the highest dose of MCh was slightly larger among subjects living in houses with (1→3)-β-D-glucan levels above 1 ng/m^3 than among those living in houses with (1→3)-β-D-glucan levels below 1 ng/m^3, but the differences were not statistically significant (−9.2%, n = 71, versus −7.7%, n = 28), with similar results being obtained when only nonsmokers and nonsmokers/nonasthmatic subjects were analyzed (data not shown). The results were similar among atopic (−9.6%, n = 14, versus −7.5%, n = 4, p = NS) and nonatopic subjects (−9.1%, n = 57, versus −7.7%, n = 24, p = NS).

Among subjects younger than 65 yr of age, there was a statistically significant inverse correlation between baseline FEV_1 and the number of years the subjects had lived in the houses investigated, when controlling for age, gender, cigarette-smoking status, asthma, atopy, and pets (β = −0.62; n = 79, p = 0.002). There was no such relationship for subjects 65 yr or older when controlling for age, gender, cigarette-smoking status, asthma, atopy, and pets (β = 0.53; n = 48, p = 0.07).

When separately analyzing male and female subjects younger than 65 yr, the analyses were limited to correlation coefficients because the number of subjects was small. Among male subjects working in environments with no exposure to organic dusts, the inverse correlation between baseline FEV_1 and the number of years lived in the house remained when controlling for age (r_{xy} = −0.57; n = 25, p = 0.003), as well as when controlling for asthma, atopy, cigarette-smoking status, and pets separately (data not shown). There was no such relationship for females (r_{xy} = −0.16; n = 45, p = 0.30).

When different levels of (1→3)-β-D-glucan were considered, the inverse relationship was present for subjects younger than 65 yr living in houses with (1→3)-β-D-glucan levels above 1 ng/m^3, when controlling for age, gender, cigarette-smoking status, asthma, atopy, and pets (β = −0.59; n = 56, p = 0.003). No inverse relationship was found for those living in houses with less than 1 ng/m^3 (1→3)-β-D-glucan.

The inverse relationship was also present for males younger than 65 yr living in houses with (1→3)-β-D-glucan levels above 1 ng/m^3 (r_{xy} = −0.52; n = 24, p = 0.009), but not for females. When separately controlling for age, cigarette-smoking status, asthma, atopy, and pets, the inverse relationship remained for male subjects (data not shown). No inverse relationship was found for those living in houses with less than 1 ng/m^3 (1→3)-β-D-glucan.

Inflammatory Markers

The amount of MPO in serum was significantly higher among subjects living in houses with (1→3)-β-D-glucan levels above 1 ng/m^3 than among those living in houses with (1→3)-β-D-glucan levels below 1 ng/m^3 (308 μg/L, n = 92, versus 261 μg/L, n = 37, p = 0.03), with similar results when only nonsmokers and nonsmokers/nonasthmatic subjects were analyzed (data not shown). The amounts of ECP and CRP in serum were slightly larger among subjects living in houses with (1→3)-β-D-glucan levels above 1 ng/m^3, but the differences were not statistically significant.

Among nonatopic subjects, the amount of MPO was also significantly higher among those living in houses with (1→3)-β-D-glucan levels above 1 ng/m^3 (310 μg/L, n = 73, versus 258 μg/L, n = 30, p = 0.03). This was not true among atopic subjects (296 μg/L, n = 17, versus 272 μg/L, n = 7, p = 0.67), but the number of atopic subjects living in houses with a low level of (1→3)-β-D-glucan was small.

Questionnaire Data

A higher proportion of subjects living in houses with (1→3)-β-D-glucan levels above 1 ng/m^3 reported symptoms of chronic bronchitis, joint pains, itchy nose, chest tightness, heaviness in the head, and unusual tiredness than did those living in houses with (1→3)-β-D-glucan levels below 1 ng/m^3. For the symptom of joint pains, the difference was statistically significant for all subjects (32%, n = 91, versus 13%, n = 37, p = 0.04). Among nonatopic subjects, a significantly higher proportion of those living in houses with (1→3)-β-D-glucan levels above 1 ng/m^3 reported symptoms of joint pains and itchy nose (36%, n = 73, versus 13%, n = 30, p = 0.02; and 13% versus 0%, p = 0.04, respectively) than did those living in houses with (1→3)-β-D-glucan levels below 1 ng/m^3. There were no significant differences among atopic subjects.

Relation to Exposure

Logistic regression analyses were performed to evaluate the relationships between (1→3)-β-D-glucan exposure and the different variables. Crude ORs as well as adjusted ORs with 95% CIs were computed while controlling for age, gender, cigarette-smoking status, pets, and atopy. The results were similar when computing crude and adjusted ORs, and thus only adjusted ORs are reported.

When controlling for age, gender, cigarette-smoking status, and pets, the OR for atopy was larger in the high-exposure group (≥ 4 ng/m^3) (OR: 2.33; 95% CI: 0.80 to 6.81), but not in the middle-exposure group (2 to 4 ng/m^3) (OR: 0.93, 95% CI: 0.24 to 3.62), as compared with the low (0 to 2 ng/m^3)-exposure group. The adjusted ORs with 95% CIs for airway responsiveness, inflammatory markers, and symptoms are shown in Table 1.

The ORs were slightly decreased with respect to CRP, and the ORs for airway responsiveness were slightly increased over the exposure range. MPO in the middle- and high-exposure groups was increased as compared with the low (0 to 2 ng/m^3)-exposure group, without a dose–response trend. The ORs for chronic bronchitis, joint pains, itchy nose, chest tightness, and heaviness in the head were larger in the two higher exposure groups (2 to 4 and ≥ 4 ng/m^3) than in low (0 to 2 ng/m^3)-exposure group, without dose–response trends.

DISCUSSION

The method for assessing levels of airborne (1→3)-β-D-glucan is a biologic test with a relatively large variation between measures. However, no other method is currently available for determining (1→3)-β-D-glucan in the low amounts dealt with in this study. The method used to produce airborne dust is aimed at replicating the dust created by normal movement in a room. The particle size was predominantly smaller than 3 μm. Other studies have sampled floor dust by vacuuming, but this does not necessarily represent the dust that is inhaled. The (1→3)-β-D-glucan levels ranged from 0 to 19 ng/m^3. Experience accumulated over the years suggests that a few nanograms of (1→3)-β-D-glucan represent normal background values, which also are present in outdoor air. Values in excess of 5 ng/m^3 are generally associated with previous mold growth or water damage. In a villa with excessive mold growth, values up to 100 ng/m^3 were recorded (25).

The method for evaluating bronchial reactivity used in the present study differs from traditional clinical testing. Instead of titrating the dose required for a certain effect (e.g., PD_{20}), a calculation was made of the average reaction to the highest cumulative dose given in a relatively short time. The same procedure for evaluating the effects of MCh challenges has been used in other, similar studies in which differences have been shown between different exposure groups (26, 27).

Atopy can be determined by different methods. The Phadiatop test used here measures the concentration of specific serum IgE antibodies against airborne allergens, which we considered to be of high relevance for this kind of study. The test is well characterized and has been used in previous studies (28, 29).

Regarding inflammatory markers, there was a slight relation between (1→3)-β-D-glucan exposure and the amount of MPO in serum, but not for the amount of ECP. In earlier studies, higher levels of ECP and MPO have been found in subjects with airway inflammation. Increased levels of MPO and ECP were found in bronchial-lavage-fluid samples from patients with chronic bronchitis (30). In a work environment with organic dusts, increased amounts of MPO and ECP in serum were found among the workers than among controls (data not published). Increased amounts of MPO and ECP in induced sputum among healthy subjects challenged with pure endotoxin (lipopolysaccharide [LPS]) were recently reported

TABLE 1

ADJUSTED ODDS RATIOS WITH 95% CONFIDENCE INTERVALS, CONTROLLING FOR AGE, GENDER, CIGARETTE-SMOKING STATUS, PETS, AND ATOPY, BETWEEN AIRBORNE (1→3)-β-D-GLUCAN, DIVIDED INTO THREE EXPOSURE GROUPS (0 TO 2 ng/m^3 AS REFERENCE), AND DIFFERENT EFFECT VARIABLES

	Glucan (*ng/m^3*)		
	0–2 OR	> 2–4 OR (CI)	> 4 OR (CI)
n	52	32	45
Continuous variables			
Baseline FEV_1	1	1.13 (0.66–1.94)	1.14 (0.73–1.78)
Airway responsiveness	1	1.00 (0.57–1.90)	1.20 (0.67–2.00)
ECP, μg/L	1	1.38 (0.87–2.16)	0.93 (0.57–1.50)
MPO, μg/L	1	1.64 (0.95–2.85)	1.32 (0.85–2.06)
CRP, μg/ml	1	0.84 (0.52–1.36)	0.74 (0.43–1.29)
Category variables			
Irritation in the eyes	1	0.91 (0.63–1.32)	0.73 (0.49–1.07)
Irritation in the throat	1	1.04 (0.70–1.55)	0.97 (0.67–1.41)
Headache	1	0.80 (0.48–1.34)	0.72 (0.43–1.20)
Irritation in the nose	1	1.23 (0.85–1.77)	0.98 (0.68–1.43)
Dry cough	1	1.05 (0.72–1.52)	1.08 (0.74–1.56)
Chronic bronchitis	1	7.99 (0.65–98.05)	2.51 (0.23–27.83)
Joint pains	1	1.55 (1.05–2.29)	1.18 (0.77–1.81)
Itchy nose	1	2.18 (0.90–5.27)	1.27 (0.62–2.58)
Chest tightness	1	2.97 (1.26–7.02)	1.35 (0.52–3.56)
Heaviness in the head	1	1.38 (0.87–2.18)	1.21 (0.78–1.88)
Unusual tiredness	1	1.11 (0.79–1.55)	0.95 (0.68–1.33)

(31). Because the exposure to airborne (1→3)-β-D-glucan in most of the 75 investigated rowhouses in the present study was rather moderate, this could explain the small and inconsistent differences in inflammatory markers between different exposure groups. It is possible that direct sampling from the airways, with induced sputum or bronchoalveolar lavage, could be a more suitable technique for detecting small changes in inflammatory markers.

The prevalence of subjective symptoms was not very high, and with few exceptions was not related to the amount of airborne (1→3)-β-D-glucan. A source of bias may have been an overreporting of symptoms among subjects living in houses with lower amounts of airborne (1→3)-β-D-glucan because of previous discussions in the area about the effects of mold growth. Furthermore, the exposure to airborne (1→3)-β-D-glucan in most of the 75 investigated rowhouses was below 10 ng/m^3, which is probably a level not sufficient to cause extensive symptoms (18).

A finding that supports the presence of airways inflammation was the inverse correlation in subjects under 65 yr of age between the baseline FEV_1 and the number of years lived in houses with higher levels of (1→3)-β-D-glucan. Theoretically, the absence of a correlation in the group exposed to low levels of (1→3)-β-D-glucan could be a consequence of the smaller number of subjects. The distribution of data was consistent over the range of years lived in these houses, however, and no trends toward a deviation from the O-line were seen. Among female subjects, no relation was found. The reason for this is unclear. Theoretically, it could be the result of differences in indoor exposures, occupational exposures, or hormone-related differences in susceptibility. The inverse relation was not found among subjects over 65 yr of age, and this may be related to the inadequate control for age in the reference material. It could also be a consequence of technical problems in measuring FEV_1 in elderly subjects.

Unexpectedly, the proportion of atopic subjects was found to be larger in the high exposure group, regardless of smoking, age, gender, pets, or asthma. This observation is based on a relatively small number of subjects and must be interpreted with caution. Theoretically, a selection bias is possible, but it is unlikely that subjects with existing atopy would preferably move to houses with higher amounts of airborne (1→3)-β-D-glucan. If true, the data suggests that sensitivity to airborne allergens can be induced by exposure to (1→3)-β-D-glucan. This hypothesis must be confirmed in further studies.

The study focused on the importance of (1→3)-β-D-glucan for airways inflammation in terms of symptoms, airway responsiveness, markers for inflammation, and atopy. Over the years, a number of substances has been suggested to be responsible for symptoms experienced in indoor air. A major such agent is house-dust-mite (HDM) allergen (32, 33). In this study, however, the houses were not particularly damp, the mold growth having occurred previously only on small areas of the building structure. Moreover, the amount of mite allergen in buildings in Scandinavia is rather low (34). Inspection of the houses did not suggest that any volatile organic compounds were involved, and there were only seven subjects exposed to environmental tobacco smoke.

Although (1→3)-β-D-glucan is a potent inflammatory agent (20, 35), the relation between exposure and effects found in this study cannot be taken as proof of causality. The responsible agent could be another substance in the mold cell that covaries with (1→3)-β-D-glucan. In view of the relationships demonstrated in this and previous studies, (1→3)-β-D-glucan can, however, be used as a marker for risk of airway inflammation.

In conclusion, the results suggest a relationship among subjects under 65 yr of age between environmental exposure to (1→3)-β-D-glucan and the proportion of atopic subjects, MPO in serum, and a decrease in baseline FEV_1 related to the number of years lived in a house containing mold.

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