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IS THE RISK OF ALLERGIC HYPERSENSITIVITY TO FUNGI INCREASED BY INDOOR EXPOSURE TO MOULDS?

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Abstract
Objectives: Moulds are frequently found in the indoor environment of residential houses. An association between domestic mould contamination and respiratory symptoms has been reported, but mould exposure as a risk factor for allergy to moulds is not well documented. The aim of the study was to evaluate the prevalence and associated factors of allergic hypersensitivity to moulds. **Materials and Methods:** A group of 243 participants was examined. Of these 118 lived in dwellings with evident signs of fungal contamination (study group) and 125 in non-contaminated sites (controls). An interview, skin prick tests to common and fungal allergens, evaluation of total serum IgE and specific IgE to moulds, resting spirometry as well as mycological analysis in building were performed for each participant. **Results:** 19.8% subjects were sensitized to at least one mould allergen. Logistic regression analysis revealed that the history of respiratory and skin symptoms, smoking cigarettes in the past and positive skin prick tests (SPT) to common allergens (dust mite and grass pollens) or the presence of a cat as a pet animal were the significant associated factors of hypersensitivity to moulds. **Conclusions:** The association between indoor fungal exposure and the development of fungal allergy was not confirmed in our study.

Key words:
Allergy symptoms, Indoor exposure, Moulds, Risk factors, Specific sensitization

INTRODUCTION

The adverse health effects of exposure to moulds are a growing public health problem. Moulds are not only common in the natural environment, but they are also frequently encountered in the indoor environment of residential houses. It is estimated that more than 400 fungal species may develop in the interior of a building [1]. *Penicillium, Aspergillus, Mucor*, *Rhizopus*, *Aureobasidium*, *Stachybotrys*, and *Cladosporium* are the most common species found in the indoor environment [2–4]. Increased airborne fungal spore concentration is often associated with musty odor, water intrusion, high indoor humidity, limited ventilation, and failure to remove indoor mould growth. It has been estimated that 25% of houses in Poland (2.7 mln houses occupied by 8 million people) are significantly contaminated with mould allergens and mycotoxins from moulds developing in the building elements and finishing materials [1].

The effect of indoor exposure to moulds on human health is highly controversial, but there are studies emphasizing that moulds growing inside dwelling houses may be the cause of many health problems [4,5]. Fungi are considered to play a role in the development of allergic airway disease and respiratory symptoms as well as various non-specific symptoms [6].

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In the present study, we analyzed the level of mould contamination in the air samples and in the samples collected from walls of the dwelling houses. The aim of the study was to evaluate the prevalence of hypersensitivity to moulds and its associated factors in the residents of mould-contaminated houses, compared with those living in the homes free from fungal contamination.

MATERIAL AND METHODS

Subjects

The study population comprised 243 persons, including 118 subjects living in 34 dwellings with evident signs of fungal contamination and 125 controls in 30 dwellings with no visible signs of such contamination. The participants were recruited between November 2004 and March 2005 from among the inhabitants of Łódź, Poland. The Regional Biomedical Ethics Committee approved the study protocol. All of the participants gave their informed consent prior to the study.

Questionnaire

The subjects were administered a questionnaire regarding respiratory, conjunctival and skin symptoms; personal and family history of atopy; tobacco smoking status; exposure to pet allergens at home; housing conditions; and history of exposure to moulds. The smoking status was denominated by three categories: active smokers, ex-smokers and non-smokers. Active smokers were defined as the participants who reported smoking cigarettes at present. Ex-smokers were those who used to smoke daily and gave up the habit at least one month prior to the survey. Non-smokers were those who had never smoked. Passive smokers were defined as non-smokers who reported sharing home with one or more smokers.

Skin prick tests (SPT)

SPT were performed on the volar part of the forearm with a standard battery of common allergens including tree and grass pollens, *Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, feathers, weeds and fungal series comprising *Alternaria tenuis*, *Aspergillus fumigatus*, *Botrytis cinerea*, *Candida albicans*, *Trichophyton mentagrophytes*, *Cladosporium herbarum*, *Fusarium moniliforme*, *Helminthosporium halodes*, *Mucor mucedo*, *Penicillium notatum*, *Pullularia pullulans*, *Rhizopus nigricans*, *Serpula lacrymans*, *Curvularia lunata*, *Phoma betae*, *Neurospora sitophila*, *Alternaria* sp., *Aspergillus* sp., *Cladosporium* sp., *Penicillium* sp., *Levures melangees*, *Charbons cerealiers* (Allergopharma, Germany). The negative control was allergen diluent and the positive one — 1 mg/ml histamine dihydrochloride solution. The largest wheal diameter was assessed after 15 min. Positive reaction was defined as a wheal diameter of at least 3 mm with no reaction to the diluent and a positive reaction to histamine [7].

Total and specific IgE

Total serum IgE was evaluated using the Uni-CAP system (Uppsala, Pharmacia Diagnostics, Sweden). Total IgE level > 100 kU/l was considered elevated.

Specific antibodies (asIgE) against fungi (*Penicillium notatum*, *Cladosporium herbarum, Aspergillus fumigatus, Candida albicans, Alternaria alternata, Helminthosporium halodes*) (mx2) were measured with the Uni-CAP system (Uppsala, Pharmacia Diagnostics, Sweden). The results were expressed quantitatively in kilo units per litre and considered positive at values higher than 0.35 kU/l.

Pulmonary function

Resting spirometry (Vicatest 2A, Mijnhardt, The Netherlands) was performed in all subjects.

Diagnostic criteria

Hypersensitivity to moulds has been defined as at least one positive result of SPT to mould allergens or a positivity of the asIgE assay.

Mycological analysis

Mould strains were isolated from the building walls and indoor and outdoor air samples. The smears from walls were collected into tubes containing 0.85% NaCl, and air samples (100 l volume) were taken using Mass Sampler (Merck) on MEA medium (Malt Extract Agar, *Oxoid*) with addition of chloramphenicol (0.1%). Additionally, sowing was performed on DG18 medium (Dichloran 18% Glycerol

Agar, Oxoid) to isolate xerophilic strains. After incubation at 27°C for 7 days, the quantity of mould growth (expressed as cfu/100 cm^2 of wall area) was determined. Mould strains were identified on the Czapek Dox Agar medium (Difco). Airborne mould concentration was expressed as cfu/m^3.

Statistical analysis

Continuous variables were expressed as mean values ± standard deviations (SD) while the nominal variables, as numbers and percentages. To identify the associated factors for developing hypersensitivity to moulds, the odds ratios (OR) and their 95% confidence intervals (CI) were calculated (EPI INFO, CDC, US). The factors that had been significant in the univariate analysis were included into the logistic regression model (Statistica'99).

The analysis of the associated factors of hypersensitivity to mould allergens concerned a group of 48 subjects who were sensitized to at least one mould allergen, and 195 non-sensitized persons. The p value below 0.05 was adopted as the reference for selecting significant associated factors.

RESULTS

Exposure assessment

High amounts of moulds were found in the indoor air as well as on the walls of dwellings with evident signs of fungal contamination (Table 1). At the sites examined, we found 14 genera of moulds, mostly *Penicillium, Cladosporium, Aspergillus, Alternaria* and *Acremonium*

Table 1. Mould contamination in dwelling houses: quantitative mycological analysis

Mould concentration	Mould contamination in buildings				Mould contamination in outdoor air N = 44
	Dwellings infected with moulds N = 34		Control dwellings N = 30		
	Walls	Indoor air	Walls	Indoor air	
Mean	7.8×10^7	3.1×10^3	3.3×10^3	2.1×10^2	5.4×10^2
Max.	8.0×10^8	2.5×10^4	3.3×10^4	7.2×10^2	1.9×10^3
Min.	2.0×10^4	1.4×10^2	1.0×10^2	4.0×10^1	8.0×10^1
SD	1.8×10^8	5.2×10^3	6.4×10^3	1.7×10^2	4.9×10^2

SD — standard deviation.

Table 2. Frequency of detecting mould growth in dwellings: qualitative mycological analysis

No	Mould species	Frequency of mould detection inside dwellings (%)				
		Infected dwellings N = 34		Control dwellings N = 30		Outdoor air N = 44
		Walls	Indoor air	Walls	Indoor air	
1	*Acremonium* sp.	13	27	13	17	32
	A. strictum	10	24	10	10	25
	A. butyrii	3	3	3	7	7
2	*Alternaria alternata*	24	33	17	23	27
3	*Aspergillus* sp.	54	75	17	20	29
	Aspergillus versicolor	36	33	10	7	11
	Aspergillus niger	12	27	7	10	9
	Aspergillus flavus	3	9	–	3	7
	Aspergillus nidulans	–	3	–	–	–
	Aspergillus ochraceus	3	3	–	–	2

Table 2. Frequency of detecting mould growth in dwellings: qualitative mycological analysis – cont.

No	Mould species	Frequency of mould detection inside dwellings (%)				
		Infected dwellings N = 34		Control dwellings N = 30		Outdoor air N = 44
		Walls	Indoor air	Walls	Indoor air	
4	*Aureobasidium* sp.	–	3	–	13	2
5	*Cladosporium* sp.	48	57	30	73	73
	Cladosporium cladosporoides	27	36	17	23	32
	Cladosporium herbarum	21	21	13	27	32
	Cladosporium sphaerospermum	–	–	–	7	5
	Cladosporium macrocarpum	–	–	3	10	2
	Cladosporium resinae	–	–	–	7	2
6	*Fusarium* sp.	–	3	–	–	2
7	*Mucor globosus*	–	6	–	7	–
8	*Paeciliomyces variotii*	–	3	–	–	–
9	*Penicillium* sp.	100	100	87	100	88
	Penicillium albidum	–	–	–	3	2
	Penicillium aurantiogriseum	9	12	3	3	2
	Penicillium brevicompactum	9	6	3	3	2
	Penicillium chrysogenum	46	42	67	77	58
	Penicillium citrinum	–	3	–	3	2
	Penicillium cyclopium	–	–	–	3	2
	Penicillium digitatum	–	3	3	3	2
	Penicillium diversum	3	3	–	–	9
	Penicillium ehinulatum	–	3	–	–	–
	Penicillium expansum	27	15	3	3	7
	Penicillium granulatum	3	6	3	–	2
	Penicillium italicum	3	3	–	–	–
	Penicillium spinulosum	–	–	3	3	–
	Penicillium terrestre	–	3	–	–	–
10	*Rhizopus nigricans*	3	9	–	10	5
11	*Scopulariopsis brevicaulis*	–	6	–	7	–
12	*Stachybotrys atra*	3	–	–	–	–
13	*Trichoderma viridie*	21	9	–	3	5
14	*Ulocladium chartarum*	3	3	–	3	2

"–" — not isolated.

(Table 2). A vast majority of mould genera occurred not only in the contaminated sites but also in the control sites and outdoor air. Two species, namely *Stachybotrys atra* and *Scopulariopsis brevicaulis* were isolated only in the contaminated areas. Several species, including *A. alternata*, *A. versicolor*, *A. niger*, *A. flavus*, *A. ochraceus*, *C. cladosporioides*, *P. expansum*, *P. aurantiogriseum*, *P. brevicompactum*, *T. viride* were more frequent in the moulds collected in the contaminated dwellings than in other environments. *P. chrysogenum* appeared to be the

most prevalent species, especially in the indoor air of the infected dwellings.

Prevalence data

The group under the study comprised 104 males and 139 females. The mean age of the subjects was 35.3 years. Lack of ventilation and mould odour were more frequently found in the sites with visible mould contamination. The study population and housing characteristics are summarized in Table 3.

The rates of the reported symptoms are displayed in Table 4. Respiratory symptoms were reported by 60.5% of the study participants (74.6% subjects and 47.2% controls). The prevalence of all symptoms was higher in the residents of mould-contaminated dwellings, but the differences were not analyzed statistically. Conjunctivitis, cough and rhinitis were most frequent in the study group. Asthma confirmed by medical diagnosis was reported by 18 subjects (7.4%). The results of SPT to common and fungal allergens as well as of evaluation of the total and asIgE are presented in Table 5.

Table 3. Characteristics of the study population

Characteristic	Total participants N = 243	Subjects N = 118	Controls N = 125
Age (mean±SD) [years]	35.3±17.6 (3; 85)	35.3±19.4 (3; 85)	35.3±15.8 (3; 76)
Sex:			
male	104 (42.8%)	50 (42.4%)	54 (43.2%)
female	139 (57.2%)	68 (57.6%)	71 (56.8%)
Smoking status:			
active smokers	40 (16.5%)	26 (22%)	14 (11.2%)
ex-smokers	65 (26.7%)	30 (25.4%)	35 (28%)
passive smokers	62 (25.5%)	42 (35.6%)	20 (16%)
Family history of atopy	98 (40.3%)	54 (45.8%)	44 (35.2%)
Type of housing:			
tenement house	71 (29.2%)	56 (47.5%)	15 (12%)
detached house	58 (23.9%)	22 (18.6%)	36 (28.8%)
apartment	114 (46.9%)	40 (33.9%)	74 (59.2%)
Housing:			
old	110 (45.3%)	62 (52.5%)	48 (38.4%)
new	133 (54.7%)	56 (47.5%)	77 (61.6%)
Lack of ventilation in the home	66 (27.2%)	50 (42.4%)	16 (12.8%)
Type of window frames:			
old wooden	124 (51%)	72 (61%)	52 (41.6%)
new plastic	99 (40.7%)	42 (35.6%)	57 (45.6%)
new wooden	20 (8.2%)	4 (3.4%)	16 (12.8%)
Mould odor detected at home	54 (22.2%)	52 (44.1%)	2 (1.6%)
Mould content in indoor air > 3×10^2	84 (34.6%)	80 (67.8%)	4 (3.2%)
Presence of *Aspergillus versicolor* or *Stachybotris atra*	46 (18.9%)	46 (38.9%)	0
Pets at home:	135 (55.6%)	59 (50%)	76 (60.8%)
dog	65 (26.7%)	36 (30.5%)	29 (23.2%)
cat	34 (14%)	21 (17.8%)	13 (10.4%)
New furniture or wall-paper at home	46 (18.9%)	30 (25.4%)	16 (12.8%)

Table 4. The prevalence of symptoms in the study population (N = 243)

Symptoms reported	Total participants N = 243	Subjects N = 118	Controls N = 125
At least one allergic respiratory symptom	147 (60.5%)	88 (74.6%)	59 (47.2%)
Rhinitis	102 (42%)	58 (49.2%)	44 (35.2%)
Dyspnea	64 (26.3%)	36 (30.5%)	28 (22.4%)
Wheezing	32 (13.2%)	20 (16.9%)	12 (9.6%)
Cough	82 (33.7%)	60 (50.8%)	22 (17.6%)
Conjunctivitis	122 (50.2%)	66 (55.9%)	56 (44.8%)
Skin symptoms	110 (45.3%)	56 (47.5%)	54 (43.2%)

Table 5. The results of SPT to common and fungal allergens and of determining total and specific IgE in the study population

Positive SPT to	Total participants N = 243	Subjects N = 118	Controls N = 125
At least one common allergen	91 (37.4%)	52 (44.1%)	39 (31.2%)
Dermatophagoides farinae	56 (23%)	28 (23.7%)	28 (22.4%)
Dermatophagoides pteronyssinus	52 (21.4%)	24 (20.3%)	28 (22.4%)
Feathers	4 (1.6%)	4 (3.3%)	0
Grass pollens	42 (17.3%)	24 (20.3%)	18 (14.4%)
Tree pollens I[1]	23 (9.5%)	12 (10.2%)	11 (8.8%)
Tree pollens II[2]	28 (11.5%)	16 (13.6%)	12 (9.6%)
Weeds	21 (8.6%)	16 (13.6%)	5 (4%)
At least one mould allergen	42 (17.3%)	24 (20.3%)	18 (14.4%)
Alternaria tenuis	20 (8.2%)	14 (11.9%)	6 (4.8%)
Aspergillus fumigatus	6 (2.5%)	6 (5.1%)	0
Botrytis cinerea	4 (1.6%)	4 (3.4%)	0
Trichophyton mentagrophytes	10 (4.1%)	6 (5.1%)	4 (3.2%)
Cladosporium herbarum	2 (0.8%)	0	2 (1.6%)
Fusarium moniliforme	0	0	0
Helminthosporium halodes	4 (1.6%)	2 (1.7%)	2 (1.6%)
Mucor mucedo	2 (0.8%)	0	2 (1.6%)
Penicillium notatum	4 (1.6%)	2 (1.7%)	2 (1.6%)
Pullularia pullulans	2 (0.8%)	2 (1.7%)	0
Rhizopus nigricans	2 (0.8%)	0	2 (1.6%)
Phoma betae	4 (1.6%)	2 (1.7%)	2 (1.6%)
Alternaria sp.	16 (6.6%)	12 (10.2%)	4 (3.2%)
Aspergillus mix.	4 (1.6%)	2 (1.7%)	2 (1.6%)
Cladosporium sp.	2 (0.8%)	0	2 (1.6%)
Penicillium mix.	6 (2.5%)	4 (3.4%)	2 (1.6%)
Basidiomycetes and yeasts			
Candida albicans	12 (4.9%)	6 (5.1%)	6 (4.8%)
Levures melanges	4 (1.6%)	2 (1.7%)	2 (1.6%)

Table 5. The results of SPT to common and fungal allergens and of determining total and specific IgE in the study population — cont.

Positive SPT to	Total participants N = 243	Subjects N = 118	Controls N = 125
Serpula lacrymans	0	0	0
Curvularia lunata	2 (0.8%)	2 (1.7%)	0
Neurospora sitophila	2 (0.8%)	2 (1.7%)	0
Charbons cerealiers	0	0	0
Total IgE (kU/l) (mean±SD) (min.; max.)	73.6±145.5 (2; 1328)	97.0±202.9 (2; 1328)	54.5±65.6 (2; 255)
IgE > 100 kU/l	56 (23%)	28 (23.7%)	28 (22.4%)
Presence of asIgE to:			
mx2[3]	16 (6.6%)	8 (6.8%)	8 (6.4%)

[1] Alder, hazel, poplar, elm, willow.
[2] Bird, beech, oak, plane.
[3] mx2 — *Penicillium notatum, Cladosporium herbarum, Aspergillus fumigatus, Candida albicans, Alternaria alternata, Helminthosporium halodes.*

Table 6. Odds ratio (OR) with 95% confidence intervals (Cl) for housing conditions in relation to hypersensitivity to mould allergens in the study population (N = 243) (univariate analysis)

Factor	Participants				OR (95% CI)
	sensitized to moulds		not sensitized to moulds		
	N	%	N	%	
Type of housing:					
tenement house	10	20.8	61	31.3	0.58 (0.25; 1.30)
detached house	12	25	46	23.6	1.08 (0.49; 2.37)
apartment	26	54.2	88	45.1	1.44 (0.73; 2.84)
Humidity at home > 60%	6	12.5	32	16.4	0.73 (0.23; 1.93)
Lack of ventilation in the home	16	33.3	50	25.6	1.45 (0.69; 3.01)
Type of window frames:					
old wooden	22	45.8	102	52.3	0.77 (0.39; 1.52)
new plastic	18	37.5	81	41.5	0.84 (0.42; 1.69)
new wooden	8	16.7	12	6.2	3.05 (1.06; 8.69)*
Housing:					
old	20	41.7	90	46.2	0.83 (0.42; 1.65)
new	28	58.3	105	53.8	1.20 (0.60; 2.39)
Visible mould contamination	24	50	94	48.2	1.07 (0.55; 2.12)
Mould odor	6	12.5	48	24.6	0.44 (0.14; 1.13)
Mould content in indoor air > 3×10^2	12	25	72	36.9	0.57 (0.26; 1.22)
Presence of *Aspergillus versicolor* or *Stachybotris atra*	6	12.5	40	20.5	0.55 (0.18; 1.44)
Pets at home:	23	47.9	112	57.4	0.68 (0.35; 1.35)
dog	12	25	53	27.2	0.89 (0.40; 1.94)
cat	14	29.2	20	10.3	3.60 (1.55; 8.37)*
New furniture or wall-paper at home	2	4.2	44	22.6	0.15 (0.02; 0.61)*
N	48		195		

* $p < 0.05$.

Forty-four percent of subjects were atopic (had at least one positive SPT) in comparison with 31% in the control group. In both groups, the positive SPT most frequently concerned D. *farinae*, *D. pteronyssinus* and grass pollens. 17.3% of the study participants were sensitized to at least one mould allergen, mainly to *Alternaria* (11.9% of subjects and 4.8% of controls). Elevated mean total IgE level was found in 23% of all

Table 7. Risk factors of hypersensitivity to mould allergens in the study population (N = 243) (univariate analysis)

Factor	Participants				OR (95% CI)
	sensitized to moulds		not sensitized to moulds		
	N	%	N	%	
Allergic disease symptoms:					
any symptoms	44	91.7	103	52.8	9.83 (3.36; 38.81)*
chronic cough	24	50	58	29.7	2.36 (1.18; 4.72)*
dyspnea	18	37.5	46	23.6	1.94 (0.94; 4.0)
wheezing	14	29.2	18	9.2	4.05 (1.71; 9.56)*
rhinitis	35	72.9	67	34.4	5.14 (2.43; 11.05)*
conjunctivitis	26	54.2	96	49.2	1.22 (0.62; 2.41)
skin symptoms	34	70.8	76	39	3.80 (1.83; 8.01)*
History of past and present disease:					
pneumonia	14	29.2	60	30.8	0.93 (0.44; 1.95)
bronchial asthma	10	20.8	8	4.1	6.15 (2.07; 18.51)*
sinusitis	4	8.3	34	17.4	0.43 (0.11; 1.31)
allergic rhinitis	24	50	22	11.3	7.86 (3.61; 17.23)*
allergic conjunctivitis	18	37.5	40	20.5	2.33 (1.11; 4.84)*
urticaria	8	16.7	38	19.5	0.83 (0.33; 2.03)
Smoking status					
active smokers	6	12.5	34	17.4	0.68 (0.22; 1.78)
ex-smokers	20	41.7	45	23.1	2.38 (1.16; 4.86)*
passive smokers	10	20.8	52	26.7	0.72 (0.31; 1.64)
Positive SPT to:					
at least one common allergen	40	83.3	51	26.2	14.12 (5.85; 35.20)*
grass pollens	26	54.2	16	8.2	13.22 (5.78; 30.64)*
tree pollens I[1]	10	20.8	13	6.7	3.68 (1.38; 9.81)*
tree pollens II[2]	14	29.2	14	7.2	5.32 (2.16; 13.16)*
weeds	6	12.5	15	7.7	1.71 (0.51; 5.02)
Dermatophagoides pteronyssinus	26	54.2	26	13.3	7.68 (3.60; 16.50)*
Dermatophagoides farinae	28	58.3	28	14.4	8.35 (3.93; 17.90)*
Family history of atopy	32	66.7	66	33.8	3.91 (1.91; 8.07)*
Total IgE > 100 kU/I	22	45.8	34	17.4	4.01 (1.93; 8.34)*
N	48		195		

*$p < 0.05$.
[1] Alder, hazel, poplar, elm, willow.
[2] Bird, beech, oak, plane.

participants. Specific IgE to fungi was found in 16 participants (6.6%).

The mean baseline spirometric values in the study group did not show significant differences in comparison with the predictive values. Mild decrease in pulmonary function was noted only in eight subjects with early recognized diseases of the respiratory system.

Associated factors of mould hypersensitivity

To investigate the relationship between housing conditions and hypersensitivity to mould allergens, logistic regression analysis was performed. Only such factors as the presence of a cat as a pet animal and the new wooden window frames correlated with allergy to moulds (Table 6).

The factors that were found to be associated with mould hypersensitivity are presented in Table 7. Chronic cough, wheezing, dyspnoea, skin symptoms, symptoms of rhinitis and conjunctivitis, hypersensitivity to common allergens and smoking in the past were associated with allergy to moulds.

Logistic regression analysis revealed that the history of respiratory and skin symptoms, smoking cigarettes in the past, positive SPT to common allergens (dust mite and grass pollens) and the presence of a cat at home were the significant associated factors of mould hypersensitivity (Table 8).

Table 8. Results of logistic regression analysis: risk factors of hypersensitivity to mould allergens. (Variables found to be significant in univariate analysis were included in the regression model)

Risk factors analyzed	p	OR (95% CI)
Allergic disease symptoms	0.003	8.06 (2.01; 32; 38)*
Skin symptoms	0.016	3.14 (1.22; 8.05)*
Presence of a cat at home	0.030	3.57 (1.12; 11.35)*
Smoking in the past	0.045	2.84 (1.02; 7.91)*
Positive SPT to at least one common allergen	0.000	8.71 (3.21; 23.65)*
Positive SPT to *Dermatophagoides farinae*	0.005	16.89 (2.37; 120.60)*
Positive SPT to grass pollens	0.000	16.62 (4.61; 59.96)*

*$p < 0.05$.

DISCUSSION

Moulds as well as house dust mite, cockroaches and pet animals are the most important indoor allergens. It has been postulated that allergy to common allergens is a risk factor of allergic diseases such as asthma, rhinitis, and eczema [8]. Nevertheless, the role of mould exposure in inducing allergic diseases is still controversial. In the present study, a survey was carried out among residents living in mould-contaminated dwellings and in the houses where no such contamination was detected. The purpose of the survey was to estimate the prevalence of allergic diseases. Due to the low specificity of a questionnaire survey, a clinical verification was performed. SPT to moulds and the determination of serum asIgE to fungi were carried out to estimate mould hypersensitivity. The usefulness of a questionnaire survey may be limited, particularly among the occupants of buildings with mould contamination. Strachan observed a discrepancy between the questionnaire and clinical data that was explained by the tenants' awareness of dampness or moulds and adverse sanitary conditions in the home [9]. Zock et al. suggested that collecting information about self-reported symptoms and mould exposure using the same questionnaire may lead to overreporting of symptoms among symptomatic subjects and underreporting among symptom-free individuals [10]. To eliminate this effect in our study, the quantity of mould growth and the selected health outcomes were measured using objective methods.

Among the microorganisms isolated in the houses with mould contamination, *A. alternata*, *C. cladosporioides*, the species known for their allergic potential, and also many species of *Penicillium* and *Aspergillus* were detected, which is in concordance with other reports [3]. Moreover, the species capable of producing mycotoxins were also isolated, these including *S. chartarum*, *A. versicolor*, *A. flavus*, *A. ochraceus*, *A. niger, P. expansum.* It was found that the main source of moulds in the air of infected dwellings were mouldy walls. However, the quality of atmospheric air can also be influenced by the composition of microbial growth inside these houses.

In our study, the average amount of moulds developing on walls in the contaminated dwellings was 1000 times as high as in the control dwellings, and the air contamination was 10 times as high. Mould content in outdoor air was

low; it was comparable to the mycological contamination in the control dwellings. It has been postulated that mould concentration in indoor air at the level of 10^3 jtk/m^3 can have a negative impact on human health [4].

An association between indoor dampness or mould contamination and respiratory symptoms has been reported [2,4,11–14]. Reports of visible growth of moulds, musty smell, damp spots and moisture also concerned correlations with eye irritation and increased rates of respiratory infections [13]. The dose-response effect was observed, i.e. an increasing level of dampness was associated with a higher prevalence of such symptoms as cough, wheezing, blocked nose and breathlessness [5]. According to Dales et al., if home dampness or mould growth is casual, it can account for 30–50% of the respiratory symptoms [15]. Exposure to mould was reported to be a significant associated factor of cold, cough, sore throat or rhinitis [13,16]. No relationship was found between chronic diseases (hypertension, angina pectoris, cancer, articular diseases) and living in moldy houses [13]. In our study, the occupants of mould-contaminated dwellings more frequently reported respiratory symptoms than did the controls (74.6% and 47.2%, respectively). For example, cough was reported by 50.8% subjects and 17.6% controls. The analysis of questionnaire data revealed that the subjects more frequently complained of pneumonia, conjunctivitis and urticaria. No other between-group differences were observed with respect to the history of past and present diseases and these findings are similar to other reports [16,17].

It is worth noting that three mechanisms of disease caused by moulds are postulated, namely, infection, allergy and toxicity, but irritation is sometimes mentioned as well [18]. In the present study, the high rate of the symptoms reported may be due to non-allergic mechanisms inducing mould-associated respiratory symptoms. Fungal metabolites such as β-glucans or mycotoxins have been suggested as an alternative mechanism of adverse effects on the respiratory system [2,10,19].

Volatile organic compounds exert an irritant effect on the respiratory mucosa [19]. In our study, *Aspergillus versicolor* and *Stachybotris atra* were found in 38.9% houses. These species may produce trichothecene that causes respiratory symptoms and skin and eye irritation.

It is postulated that an increased exposure to indoor allergens is an important risk factor of developing asthma and allergic sensitization [20]. Gunnbjornsdottir et al. recently reported that people living in damp houses had a higher prevalence of respiratory symptoms and asthma [6]. Kilpeläinen et al. also found a strong association between exposure to visible mould and asthma [12]. In the present study, bronchial asthma was reported twice as much frequently in the group of subjects than controls (10.2% vs. 4.8%). On the other hand, the prevalence of asthma at the level of 10.2% is comparable to that observed in the general population [10]. For comparison, Garrett et al. did not observe any association between spore concentration and respiratory symptoms [2]. In the study by Taskinen et al., the prevalence of asthma among children from the schools with moisture problems and among controls was similar, whereas the respiratory symptoms like wheezing and cough were more frequent in mould-contaminated schools [21]. It is worth noting that mould exposure among subjects sensitized to moulds and those with bronchial asthma, allergic rhinitis or atopic dermatitis, intensified the symptoms and worsened the course of disease. Zock et al. reported that moisture and the presence of mould in the home were related to asthma symptoms and bronchial responsiveness, and the association of mould exposure with asthma was stronger among mould-sensitized subjects than non-sensitized individuals [10].

It is postulated that indoor allergen exposure may lead to specific sensitization. In our study, 37.4% of the study population had positive SPT to common allergens (44.1% of subjects and 31.2% of controls). Thus, the prevalence of atopy is similar to that in the general population [22]. In the study by Garrett et al., positive reactions to extracts in skin prick testing were more common at higher levels of contamination with *Cladosporium* or *Penicillium*. Besides, exposure to *Aspergillus* was found to be a risk factor for atopy [2].

The problem of exposure to moulds as a factor inducing hypersensitivity is still controversial. It is suggested that mould exposure may initiate IgE reactions but there is little evidence of such allergy among inhabitants of mouldy homes. In our study, allergy to moulds was found in 17.3% of the study participants (20.3% subjects and 14.4% controls). It is estimated that at least 3–10% of adults and

children have positive SPT to fungal allergens [23]. As in other studies, *Alternaria tenuis*, *Candida albicans* and *Trichophyton* were found to be the most frequent sensitizers (in 8.2%, 4.9% and 4.1% of participants, respectively) [24]. Apart from the hypersensitivity to *Alternaria* sp. (11.9% vs. 4.8% sensitized), no other between-group differences could be found. On the other hand, it is important to note that in the present study, an increased level of *Alternaria* spores was detected only in two dwellings.

Pappas et al. found that sensitization to moulds was more common in damp housing [19]. Fungal allergies were more frequent among children exposed to *Cladosporium* or *Penicillium* or to musty odour [2]. For comparison, Dotterud et al. did not find any association between mould contamination in the home and specific sensitization [3]. Dharmage et al. reported that high exposure to airborne fungi was associated with increased bronchial hyperreactivity; however, paradoxically, also with a lower risk of mould sensitization [25]. Likewise, the levels of exposure to mould allergens, investigated under the Childhood Asthma Management Program, were not found to be related to sensitization rates [26].

In our study, serum specific IgE to moulds was found in 6% of participants in both groups. This finding is consistent with previous reports [10, 23]. Burr et al. described more frequent occurrence of specific IgE to *Penicilium notatum* among asthmatic inhabitants of moldy homes than among controls and among asthmatics from homes with no mould contamination [27]. However, our study did not confirm these findings.

In the present study, a significant correlation was found between the history of respiratory and skin symptoms, smoking cigarettes in the past, positive SPT to common allergens, presence of a cat at home and the hypersensitivity to moulds. Our study confirms the rare occurrence of isolated mould hypersensitivity which usually coexists with allergy to other aeroallergens [18]. It has been found that the presence of a cat or dog as a pet animal increases fungal population in the home and, therefore, may promote allergy to moulds [28]. Atopy was regarded as a factor predisposing to mould allergy [17]. It is not explained how tobacco smoking can affect the hypersensitivity to moulds. Tariq et al. found that passive smoking had no influence on mould sensitivity [29]. However, there are data indicating that smoking is associated with an increased risk of sensitization to house dust mite but a decreased risk of sensitization to grass pollens and cat allergens [30]. Interestingly, in our study, neither living in mould-contaminated dwellings with no ventilation and high humidity, nor exposure to *Aspergillus versicolor* or *Stachybotris atra* present in indoor air were found to be the associated factors of mould hypersensitivity.

CONCLUSIONS

1. Among the inhabitants of mould-contaminated houses, the hypersensitivity to moulds is similar to that observed in the general population.
2. A history of respiratory and skin symptoms, smoking cigarettes in the past, positive skin prick tests to common allergens (dust mite and grass pollens) and the presence of a cat at home are the significant associated factors of mould hypersensitivity.
3. Fungal exposure in damp home environment was not found to be a significant risk factor for developing hypersensitivity to mould allergens.

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