
Characterization of a population presenting with suspected mold-related health effects

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Background: Numerous epidemiologic studies have reported a relationship between dampness and increased respiratory symptoms in adults and children, which has led to increasing concerns among the general population about potential mold-related health effects.

Objective: To identify characteristics of patients with possible mold-related health effects that might help allergists better understand how to evaluate such patients in their practices.

Methods: The parameters that were evaluated for the total population included age, sex, upper and lower respiratory tract symptoms, systemic symptoms, atopic status, pet ownership, smoking history, mold sensitization, mold exposure, and exposure location. Subgroups stratified by atopic status, mold sensitization, and indoor mold exposure confirmed by an environmental mold report prepared by certified industrial hygienists were also analyzed. Significant associations were determined by correlation coefficients and logistic regression analysis.

Results: A total of 135 patients with possible mold-related health effects secondary to prolonged indoor mold exposure were evaluated. For the overall population, a strong correlation was found among atopy, mold sensitization, and sensitization to specific molds identified in the patient's environmental report. Mold intracutaneous testing was not useful for discriminating between atopic and nonatopic individuals. There were no associations among the patients' presenting symptoms, atopic status, and magnitude of mold exposure. Similar findings were identified when subgroup analysis was performed.

Conclusions: Patients who present with mold-related health effects manifest heterogeneous characteristics. Medical history, skin testing, and current indoor mold sampling practices have significant limitations in establishing a causal link among mold exposure health effects.

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INTRODUCTION

Fungi have been implicated in causing allergic rhinitis, asthma, and infection, and their byproducts are known to induce both toxic and irritant effects.¹ Current mold-related issues are primarily due to indoor water intrusion, which creates a moist environment conducive for mold growth. Public concerns for mold-related health effects have recently increased because of media attention given to several epidemiologic studies that address indoor mold and potential health problems in children and adults.^{2–5}

Epidemiologic studies conducted worldwide that used questionnaire surveys and/or home inspection to investigate home dampness have found a wide prevalence ranging from 9% to 59%.^{2–7} The 2001 Nordic Interdisciplinary Review of Scientific Evidence on Associations Between Exposure to “Dampness” in Buildings and Health Effects conducted a literature review to investigate the relationship between dampness and health effects.⁸ In damp homes, there was an increased odds ratio (between 1.4 and 2.2) for cough, wheeze, asthma, airway infections, fatigue, and headaches.⁸ Based on

these findings, it was concluded that there was sufficient evidence to warrant preventive measures against home dampness.⁸ The 2002 European Community Respiratory Health Survey examined the association among housing characteristics, mold and dust mite levels, and adult asthma, using questionnaire surveys, specific IgE testing, and objective lung function studies.^{9,10} Questionnaire data obtained in 19,218 patients and health data collected in approximately 15,202 patients indicated that indoor mold growth had an adverse effect on adult asthma.^{9,10}

Other studies have reported an increased association between childhood asthma and living in damp homes.^{11–13} Although these studies consistently report strong associations between dampness and respiratory symptoms, objective markers of asthma, such as pulmonary function, are often not confirmatory.^{12,13} Furthermore, questionnaires are often inadequate for predicting the presence of increased indoor mold exposure, and air sampling is too variable for establishing a relationship among indoor mold growth, mold sensitization, and clinical symptoms.^{14–16} These limitations have made it virtually impossible to set guidelines for indoor fungal levels that can be used to predict risks for mold sensitization and clinical symptoms. Other confounding variables, such as other indoor allergens and/or irritant or toxic exposures, known to have an impact on health must also be factored into

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a cause-and-effect relationship between indoor mold exposure and adverse health effects.¹⁷

Patients who present with mold-related health effects often are poorly characterized, and little is known about short- and long-term outcomes in this population. It is important that physicians recognize the predictive potential of specific clinical features so they can correctly provide advice on whether mold exposure is related to their symptoms and, if so, determine whether it poses risks for long-term health problems. This study identifies characteristics of patients with possible mold-related health effects that might help allergists better understand how to evaluate such patients in their practices.

METHODS

A retrospective medical record review with longitudinal follow-up was conducted in patients who presented with mold-related health effects in homes or workplaces between January 2001 and January 2002. All patients received a thorough physician-administered history, which included a detailed home and workplace environmental history, physical examination, and prick-puncture (PP) skin testing followed by intracutaneous testing and/or specific IgE serologic testing to seasonal and perennial allergens, including a panel of mold allergens. All patients also completed an environmental housing characteristic questionnaire, which was used to validate responses obtained by physician-administered history. Spirometry was performed when clinically indicated. A database was generated by inclusion of demographic characteristics (age and sex) of each patient, the presence of upper or lower respiratory tract symptoms, systemic symptoms, P/P skin testing, and intracutaneous testing and/or specific IgE tests to seasonal and perennial allergens (including mold allergens). Indoor environmental mold reports prepared by certified industrial hygienists were obtained when available. The mold panel included *Alternaria alternata*, *Aspergillus fumigatus*, *Aspergillus niger*, *Cladosporium herbarum*, *Cladosporium sphaerospermum* (1:40 wt/vol), *Candida albicans*, *Curvularia spicifera* (1:40 wt/vol), *Epicoccum purpurascens* (1:40 wt/vol), *Fusarium moniliforme*, *Bipolaris sorokiniana*, *Mucor mucedo*, *Penicillium notatum*, *Phoma betae* (1:40 wt/vol), *Rhizopus nigricans* (1:40 wt/vol), *Stemphylium solani* (1:40 wt/vol), *Trichophyton rubrum*, and *Stachybotrys chartarum* (Greer Laboratories, Lenoir, NC). The mold antigen concentrations were 1:20 (wt/vol) for P/P skin tests and 1:1,000 for intracutaneous tests, unless otherwise specified.

Symptom categories were divided into upper respiratory tract, lower respiratory tract, and systemic based on all of the presenting symptoms of this patient population. Symptoms were then grouped within each category according to correlation coefficients calculated to compare individual symptoms with one another. Involvement of the upper airway was defined as having 1 or more symptoms of nasal congestion, postnasal drainage, headaches, ear plugging, runny nose, sneezing, or itching of the eyes, ears, nose, and throat. Lower respiratory tract problems were defined as having 1 or more symptoms of wheezing, coughing, chest tightness, or short-

ness of breath. The systemic category was defined as having 1 or more symptoms of cognitive impairment, rash, or joint pains. A diagnosis of asthma was defined as a change in forced expiratory volume in 1 second (FEV₁) of 12% or greater and/or a provocation dose of methacholine that caused a decrease in FEV₁ of 20% at 10 mg/mL or lower. Patients were considered atopic if they exhibited 2 or more positive P/P skin test results, manifested as a wheal size of 3 mm or larger with flare compared with a negative saline control. Positive histamine controls (10 mg/mL) were also included in the test panel.

The parameters analyzed included age, sex, site of mold exposure, passive smoke exposure, indoor pets, age of the home, upper respiratory tract symptoms, lower respiratory tract symptoms, systemic symptoms, atopic status (defined as 2 positive P/P skin test results to seasonal and/or perennial allergens), the presence of 2 or more positive intracutaneous tests to seasonal and/or perennial aeroallergens (excluding molds), 2 or more positive mold P/P skin test results, 2 or more positive mold intracutaneous test results, the presence of 2 or more positive mold P/P skin test results to which the individual was exposed based on an environmental evaluation, and 2 or more intracutaneous molds to which the individual was exposed based on an environmental evaluation. A subanalysis was also performed on those patients who presented with an indoor environmental evaluation report, focusing on skin test results specific to the molds identified in their environment.

In those individuals who presented with an indoor environmental exposure report, a 9-point mold exposure scale was used to correlate the severity of mold contamination in their environments with these clinical factors. Table 1 summarizes the criteria for the quantitative mold exposure scale used in this analysis. Individuals with exposure scores of less

Table 1. Nine-Point Mold Exposure Scale*

Exposure criteria	Points
Total mold spore count indoor greater than outdoor	1
Differences in indoor vs outdoor mold species	1
Percentage of <i>Aspergillus</i> or <i>Penicillium</i> species indoor greater than outdoor	1
Presence of <i>Stachybotrys</i> in any indoor samples	1
Percentage of <i>Aspergillus versicolor</i> indoor greater than outdoor	1
Wall cavity sample >5,000 CFU/m ³	1
Bulk dust sample >25,000 CFU/g	1
Total air sample, CFU/m ³	
< 500	0
500–1,000	1
> 1,000	2

* Criteria in this scale are regularly used by certified industrial hygienists to determine whether indoor mold exposure is a problem. These criteria are not standardized, and this scale had not been validated.

than 3, 3 to 6, and more than 6 were designated as having low, moderate, and high mold contamination, respectively.

Calculation of correlation coefficients and subsequent logistic regression analysis for the defined parameters were performed using SAS statistical software (SAS Institute Inc, Cary, NC). $P < .05$ was considered statistically significant.

RESULTS

A total of 135 patients with possible mold-related health effects secondary to prolonged indoor mold exposure were evaluated. Table 2 summarizes the demographic characteristics of the total population and pertinent subpopulations. Patient ages ranged from 1 to 71 years. Forty-one percent were male, with 96% white and 4% African American. There were 22 homes where more than 1 individual was evaluated under the same roof, comprising 63 total individuals. Therefore, the total population represented a total of 92 different home and 2 workplace environments. Ninety-five percent of patients were self-referred or referred by their primary care physician based on recommendations by an industrial hygienist (M.S.C.) to evaluate potential mold-related health effects. Eighty percent of these individuals had an attorney at the time of their initial evaluation (10 different plaintiffs, 3 different defense attorneys) and 20% had no attorney. Five percent came for evaluation without recommendations from either an industrial hygienist or an attorney. Only 2 patients were seeking worker's compensation; the remaining 133 cases were related to mold exposure in the home. Of this population, 40 individuals were diagnosed as atopic (see the "Methods" section), and 70 patients had nonallergic or irritant-induced rhinitis. Ninety percent of the patients underwent P/P skin testing to evaluate sensitization to seasonal and perennial allergens, including the mold panel, whereas 7% underwent serologic testing because skin testing could not be performed due to dermatographia or the use of underlying medications that could not be discontinued. Only 1 patient underwent both serologic and skin testing, the results of which were both negative, indicating a good intertest correlation for this individual. Eighteen patients had objective criteria consistent with asthma (see the "Methods" section), among whom 16 were atopic. Sixty-six patients had an indoor environmental evaluation previously performed by a certified industrial hy-

gienist. Some parents refused skin and/or serologic allergen testing of their children, and spirometry was generally not possible in children younger than 6 years.

For the overall population ($n = 135$), there was a strong association between being sensitized to seasonal and/or perennial aeroallergens and mold skin sensitization (Table 3, $P < .001$). Atopic patients were more likely to manifest skin sensitivity to molds that were identified in their environmental survey ($P < .001$). It should be emphasized that it was not possible to determine when and where mold sensitization occurred in this population. Surprisingly, these same individuals exhibited a higher FEV₁ ($P < .01$) and had fewer lower respiratory tract symptoms compared with nonatopic individuals ($P < .04$). Also noteworthy was the fact that pet owners and smokers were less likely to be atopic ($P = .05$ and $P < .01$, respectively) and cat owners were less likely to show skin sensitization to molds ($P < .04$). None of the patients, regardless of atopic status, elicited a positive skin prick test result to *Stachybotrys*. A similar analysis of the smaller atopic population ($n = 40$) demonstrated similar correlations ($P < .05$).

Nonatopic patients ($n = 70$) were more likely to experience upper and lower respiratory tract symptoms compared with the atopic population ($P < .001$). Patients who complained of systemic symptoms were more often likely to be nonatopic ($P < .05$) and living in older (>30 years old) homes ($P < .001$). For both the atopic and nonatopic populations, there was a strong correlation between positive mold intracutaneous testing and indoor exposure to the same mold allergens ($P < .001$). This could be interpreted to mean that intracutaneous testing is not useful as a means of discriminating between atopic and nonatopic phenotypes, because the intracutaneous concentration was too high in this study. All of the statistically significant correlations found for the total atopic and nonatopic populations also pertained to the subanalysis of those individuals, whether atopic or nonatopic, who had indoor environmental evaluation reports ($n = 66$).

Correlation of mold severity exposure using the 9-point scale (Table 1) with the clinical parameters named herein did not distinguish between these populations. It is likely that patients who hired an industrial hygienist to conduct an indoor mold analysis had strong reasons to suspect a mold

Table 2. Demographic Characteristics of the Patient Populations Evaluated

Population	Mean age, y	Median age, y	Maximum age, y	Male, No.	Female, No.	African American, No.	White, No.
Total ($n = 135$)	33	35	71	56	79	5	130
Nonatopic ($n = 70$)	33	36	71	20	50	2	68
Atopic ($n = 40$)	34	36	62	21	19	3	37
Mold report ($n = 66$)	30	33	58	33	33	1	65
Atopic with mold report ($n = 22$)	33	34	52	12	10	1	21
Nonatopic with mold report ($n = 36$)	31	36	58	24	12	0	36

Table 3. Summary of Significant Correlations between Parameters Analyzed (n = 135)

Parameters	Atopic (n = 40) vs nonatopic (n = 70)*	R value†	P value	Description
No. of positive P/P skin test results vs No. of positive P/P skin test results to mold	Atopic	0.4	<.001	Atopic patients were more likely to be sensitized to mold allergens
No. of positive P/P skin test results vs No. of positive P/P skin test results to molds present indoor	Atopic	0.4	.003	Atopic patients were more likely to be sensitized to mold allergens identified in their environment
No. of positive P/P skin test results to molds vs No. of positive P/P to molds present indoor	Atopic	0.8	<.001	Patients sensitized to mold allergens were more likely to be exposed to these specific mold allergens in their environment
No. of positive P/P skin test results to molds vs lower respiratory tract symptoms	Atopic	−0.2	.04	Patients sensitized to mold allergens were less likely to experience lower respiratory tract symptoms
No. of positive intracutaneous test results positive to molds vs No. of intracutaneous test results positive to molds present indoor	Atopic/nonatopic	0.7	<.001	Patients with positive mold intracutaneous testing were more likely to be exposed to these specific mold allergens in their environment
No. of P/P skin test results positive to molds present indoor vs lower respiratory tract symptoms	Atopic	−0.2	.05	Patients sensitized to mold allergens that they are exposed to indoor are less likely to experience lower respiratory tract symptoms
Upper respiratory tract vs lower respiratory tract symptoms	Nonatopic	0.3	<.001	People with upper respiratory tract symptoms also have lower respiratory tract symptoms.
Indoor pets vs No. of positive P/P skin test results	Nonatopic	−0.2	.05	People with pets are less likely to be atopic
Indoor smoke exposure vs No. of positive P/P skin test results	Nonatopic	−0.3	.01	Smokers are less likely to be atopic
Indoor cats vs No. of P/P skin test results positive to molds	Nonatopic	−0.2	.04	People with cats were less likely to be sensitized to molds.
PFT vs mold P/P skin test	Atopic	0.4	.01	Patients sensitized to mold have higher pulmonary function test results
PFT vs IEAR P/P skin test	Atopic	0.4	.02	Patients sensitized to molds present indoors have higher spirometry values

Abbreviations: IEAR, indoor environmental assessment report; PFT, pulmonary function testing; P/P, prick-puncture.

* Significant for atopic and/or nonatopic subgroups.

† Negative values reflect an inverse correlation; positive values reflect a direct correlation.

problem due to the presence of visible mold and/or mildew. Therefore, most homes investigated under these circumstances would be biased toward a high mold exposure scale score.

All of the patients investigated in this study were followed up prospectively for at least 2 years. Most of these individuals either changed their living or work environment or implemented proper remediation to improve their indoor air quality. Most (99%) had partial or complete elimination of their symptoms after these interventions. Thus far, there have been only 2 patients with probable serious health effects related to indoor mold exposure observed in this population. However, only 1 had health problems that could be adequately linked to mold exposure.¹⁸ The remaining individuals diagnosed as having allergic or nonallergic rhinitis and/or asthma have been well controlled with avoidance measures and/or controller pharmacologic therapy. In most cases, it was possible to demonstrate that the indoor environment aggravated their rhinitis and/or asthma symptoms, but establishing a cause and effect was more problematic despite applying algorithmic assessments similar to those used for the diagnosis of occu-

pational asthma. Most cases are still in litigation with an employer, builder, contractor, or insurance company. Several individuals presented with situational anxiety and depression related to their ongoing litigation and concerns about potential mold-related health effects. However, none of these individuals had been diagnosed as having an underlying major affective disorder (depression, bipolar, schizophrenia) before becoming concerned that indoor mold exposure may be contributing to their health-related problems.

DISCUSSION

Several results from this study are noteworthy. In evaluating the total population, atopic patients were more likely to be sensitized to mold allergens that were more likely to be found in air sampling analysis of their indoor environments. A similar correlation was found in atopic patients (n = 40). Surprisingly, patients with mold skin sensitivity were less likely to complain of lower respiratory tract symptoms and had better lung function compared with nonatopic individuals. The poor correlation of objective lung function with mold exposure and sensitization has been previously reported in the

literature.^{12,13} This observation emphasizes the importance of correlating clinical history with skin test sensitization and other objective testing to establish a diagnosis of allergies and/or asthma.

Intracutaneous testing (1:1,000 dilution) did not discriminate between atopic and nonatopic phenotypes, similar to what has been widely reported.¹⁹ If positive intracutaneous test results were used to define atopy, both atopic and nonatopic populations had positive intracutaneous test results to molds they were exposed to indoors, whereas when the P/P skin test criteria were used, only atopic patients were skin test sensitive to those molds in their indoor environments. Therefore, it is possible that mold intracutaneous testing at a 1:1,000 dilution could lead to irritant reactions and erroneous conclusions regarding the mechanism for a patient's clinical symptoms. However, this finding might also indicate that intracutaneous testing represents a surrogate marker for mold exposure. In the future, intracutaneous threshold titration mold skin testing studies should be conducted to better differentiate between irritant and immune-mediated responses and to establish whether this test could be used as a possible clinical marker for mold exposure.

Other notable findings were that pet owners and smokers were less likely to be atopic and cat owners were less likely to be sensitized to molds. These findings require confirmation in a larger study that incorporates a cohort control population in its design. Recent studies have reported that children born into homes with either a cat or dog are less susceptible to becoming sensitized.^{20,21} Whether this reflects an allergen dose-response relationship that leads to tolerance vs sensitization or is the result of population bias in that owners of pets are less likely to be atopic requires further investigation.^{22,23} Conflicting results regarding the relationship between smoking and atopy has also been reported in previous studies. However, the inverse relationship between smoking and atopy in our population is supported by a large cross-sectional study conducted in Sweden with almost 7,000 young and middle-aged adults (ages 16–49 years) and almost 4,500 of their children (ages 3–15 years), which found a low risk for atopic disorders in smokers and their children.²⁴

A major limitation of this study was population bias. All patients in this study were specifically referred because of suspected health problems related to mold exposure. To minimize this problem, all clinical data were entered into a database without patient identifier information and analyzed according to precise criteria established at the onset of the study. Because this study was a retrospective medical record review with longitudinal follow-up of patients who present with suspected mold-related health effects, it was not readily possible to identify a control cohort population with which to compare our findings due to lack of adequate financial support.

This study also attempted to correlate mold-related health effects with exposure severity. This was not possible, because analysis of the reports from all of the homes or workplaces resulted in high scores. This is likely because all of the

individuals with an indoor environmental evaluation report lived or worked in environments with visible mold. However, it is also possible that the criteria used to define mold exposure severity were set too low, making the scale too sensitive. Further blinded randomized sampling studies of homes are needed to determine the specificity and sensitivity of normal ranges for indoor mold exposure.

In summary, retrospective analyses of patients who present with mold-related health effects is a useful approach to characterizing patients with mold-related complaints. Although the current data are useful for generating hypotheses, it is clear that large, prospective studies with appropriate control populations are necessary to more clearly understand the issues pertaining to mold exposure in the home and workplace. Future studies will have to be carefully designed to control for confounding variables that have previously made it difficult to correlate mold exposure with clinical symptoms.

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