
Allergy and “toxic mold syndrome”

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Background: “Toxic mold syndrome” is a controversial diagnosis associated with exposure to mold-contaminated environments. Molds are known to induce asthma and allergic rhinitis through IgE-mediated mechanisms, to cause hypersensitivity pneumonitis through other immune mechanisms, and to cause life-threatening primary and secondary infections in immunocompromised patients. Mold metabolites may be irritants and may be involved in “sick building syndrome.” Patients with environmental mold exposure have presented with atypical constitutional and systemic symptoms, associating those symptoms with the contaminated environment.

Objective: To characterize the clinical features and possible etiology of symptoms in patients with chief complaints related to mold exposure.

Methods: Review of patients presenting to an allergy and asthma center with the chief complaint of toxic mold exposure. Symptoms were recorded, and physical examinations, skin prick/puncture tests, and intracutaneous tests were performed.

Results: A total of 65 individuals aged 1½ to 52 years were studied. Symptoms included rhinitis (62%), cough (52%), headache (34%), respiratory symptoms (34%), central nervous system symptoms (25%), and fatigue (23%). Physical examination revealed pale nasal mucosa, pharyngeal “cobblestoning,” and rhinorrhea. Fifty-three percent (33/62) of the patients had skin reactions to molds.

Conclusions: Mold-exposed patients can present with a variety of IgE- and non-IgE-mediated symptoms. Mycotoxins, irritation by spores, or metabolites may be culprits in non-IgE presentations; environmental assays have not been perfected. Symptoms attributable to the toxic effects of molds and not attributable to IgE or other immune mechanisms need further evaluation as to pathogenesis. Allergic, rather than toxic, responses seemed to be the major cause of symptoms in the studied group.

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INTRODUCTION

“Toxic mold syndrome” refers to a constellation of nonspecific symptoms in patients exposed to mold-contaminated environments. Symptoms include headache, rhinorrhea, cough, dyspnea, epistaxis, diarrhea, nausea, vomiting, memory loss, generalized and focal weakness, and mood changes. Mold exposure has been implicated in exacerbating asthma and hypersensitivity pneumonitis in susceptible individuals.^{1,2} Thus, the health effects of mold exposure may be variable and include IgE- and other immunologically mediated hypersensitivity diseases and symptoms that may not be mediated by immune mechanisms. There is a lack of objective evidence linking mold identified in homes and the nonspecific symptoms of the exposed patient. This void makes toxic mold syndrome controversial. Numerous case reports and small case-control studies^{3–6} have implicated water damage, growth of the mold *Stachybotrys* species, and nonspecific symptoms. Idiopathic pulmonary hemorrhage in at least 100 infants associated with exposure to *Stachybotrys* species has also been reported; however, this association has been questioned.^{7–11} It is the perception of the adverse effects of this

organism that gives the constellation of symptoms the name *toxic mold syndrome*.

An association between “damp” environments and health effects has been established.¹² The etiology of the health effects has yet to be identified. Possible mechanisms include IgE-mediated, mycotoxin-related, direct irritation by mycelial components, volatile organic compounds (VOCs), or immune responses, inducing asthma or hypersensitivity pneumonitis. In another mold-related illness, “sick building syndrome,” there is controversy regarding the etiology because no single identifiable factor has been established as causing the nonspecific symptoms seen in this illness. Theories regarding etiology have centered on microbial VOCs, airborne biological endotoxins, and psychological issues. Most important, inadequate ventilation has been a key component to the sick building syndrome hypothesis.¹³

To evaluate these associations, we performed a retrospective study of patients who presented to a tertiary care asthma and allergy center with chief complaints of health symptoms related to environmental mold exposure as a result of water incursion into the home, work, or school environment leading to mold amplification. The objective of this study was to evaluate the patients’ signs and symptoms and the presence of atopy to characterize the clinical features and define the etiology of their complaints. We also reviewed mold survey data from patients’ work, school, and home environments when available. This study was approved by the human rights

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PATIENTS AND METHODS

We reviewed 65 patients who attended the Asthma and Allergy Center of the Medical College of Wisconsin with the chief complaint of health changes believed to be the result of mold exposure. Specifically, we reviewed symptoms, medical history, medication use, family history, social history (which included smoke and pet exposure), and physical examination findings. In addition, we reviewed a standard intake questionnaire regarding mold exposure in the home, school, or office. If available, mold surveys of affected environments were reviewed. Patients presented from all around the United States; thus, we could not conduct our own environmental surveys. As a result, several different industrial hygienists who were not using identical techniques performed the mold surveys, but we report what was completed in the "field," as provided by the patients.

Allergy skin prick/puncture tests (SPTs) or, where appropriate, intracutaneous tests (ICTs) were performed on patients without contraindications. Adults were tested with 39 molds by SPT and, if necessary, 5 ICTs composed of mixtures of eight 1:500 dilutions of each of the molds. The ICTs were performed in the event of negative SPT results. These mixtures were shown not to cause irritation after injection, and they were used to avoid performing up to 39 ICTs. Testing was deferred in 3 patients (1 was pregnant and 2 refused testing). Appropriate negative (saline) and positive (histamine) controls were used. Children were tested with 8 to 15 select molds via SPT and, where feasible, with the ICT solutions. Children underwent fewer SPTs because we considered that it was not ethical to perform 39 SPTs when we could obtain appropriate clinical data regarding general mold allergy with fewer tests. Ragweed, tree, grass, mites, dog, and cat were also tested via SPT or ICT in all patients when

possible. A positive SPT result was defined as a wheal diameter 3 mm greater than the negative control. The mold extracts (Antigen Laboratories Inc, Liberty, MO) were administered using the Quintip skin test device (Hollister-Stier Laboratories LLC, Spokane, WA) for SPTs and via 27-gauge needles using separate syringes for ICTs.

RESULTS

A total of 65 patients, including 13 families (defined as ≥ 2 people; some people came alone), were evaluated. Twenty-nine patients (45%) were male and 36 (55%) were female. Their ages ranged from 1½ to 52 years. The characteristics of the study group are summarized in Table 1. The reported sites of mold exposure included homes (69%), schools (22%), and office buildings (9%). Twenty-two percent (14/65) of the patients had a previous physician diagnosis of asthma, and 14% (9/65) had a previous diagnosis of allergic rhinitis. Eighty-one percent (52/64) of the patients had pets in and around the home, including dogs, cats, hamsters, mice, rats, birds, rabbits, goats, chinchillas, and horses. Fifty percent (32/64) of the patients were either smokers or were exposed to environmental tobacco smoke on a long-term basis. Fifty-four percent (35/65) of the studied population (8 of 13 families) had a mold survey of their home, school, or work environment. Evaluation of the mold surveys revealed a predominance of *Aspergillus*, *Penicillium*, and *Cladosporium* obtained from air and surface samples. As noted previously herein, the survey techniques were not standardized because the results were obtained by varied environmental investigations around the country.

The presenting symptoms in order of predominance were rhinitis (congestion and discharge) (62%), cough (52%), headache (34%), respiratory symptoms (including dyspnea, wheeze, chest tightness, or shortness of breath) (34%), ocular pruritus (29%), central nervous system (CNS) symptoms (including dizziness, anxiety, weakness, restless legs, mem-

Table 1. Demographic Characteristics of the Patient Population*

	Children (n = 36)	Adults (n = 29)	Total (N = 65)
Sex			
M	16 (44)	13 (45)	29 (45)
F	20 (56)	16 (55)	36 (55)
Age, mean (range), y	8 (1½–17)	37 (18–52)	21 (1½–52)
Medical history			
Asthma	8 (22)	6 (21)	14 (22)
Allergic rhinitis	3 (8)	6 (21)	9 (14)
Mold exposure site			
Home	23 (64)	22 (76)	45 (69)
School	13 (36)	1 (3)	14 (22)
Work/office	0	6 (21)	6 (9)
Social history†			
Smoke exposure	19 (53)	13 (45)	32 (50)
Pets	29 (81)	23 (79)	52 (81)

* Data are given as number (percent) of patients, except where indicated otherwise.

† N = 64 for social history.

ory loss, and shaking) (25%), fatigue (23%), gastrointestinal symptoms (including nausea, vomiting, and abdominal pain) (17%), epistaxis (6%), and dysuria (5%) (Table 2).

Forty-two percent (27/65) of the patients had normal physical examination findings. The remaining patients had findings suggesting allergic disease, including pale nasal mucosa, turbinate edema, "cobblestoning" of the posterior pharynx, cervical lymphadenopathy, allergic "shiners," and nasal discharge. Of 14 patients with asthma in the study population, 12 had normal pulmonary function test results and 2 had mild or moderate obstructive function.

Fifty-three percent (33/62) of the patients had positive SPT or ICT reactions to molds (Table 3); 40% (22/55) of the patients had positive SPT or ICT reactions to other environmental allergens only (including trees, grasses, ragweed, dog, cat, and mites but not including mold) (Table 4). Sixty percent (37/62) of the patients had positive SPT or ICT reactions to at least 1 allergen (including molds, mites, trees, ragweed, grasses, dog, and cat). Thirty-three percent (18/55) of the patients had positive SPT or ICT reactions to both molds and other environmental allergens (Table 4). Fifty-seven percent (8/14) of the patients with asthma had positive SPT or ICT reactions to molds, and 89% (8/9) of the patients with a history of allergic rhinitis had positive SPT or ICT reactions to molds (Table 4). Of the 53% (33/62) of the patients who had a positive SPT or ICT reaction to molds, 24% (8/33) were positive only on ICT with the mold mixtures. Sixty-one percent (11/18) of the patients had positive mold SPT or ICT reactions that correlated with the species of mold found during inspection of their environments. Of the patients with a positive SPT or ICT reaction to molds, 88% (28/32) were pet owners.

DISCUSSION

Patients presented to the Asthma and Allergy Center complaining of toxic mold exposure; we retrospectively evaluated and characterized the etiology of their symptoms using his-

tory, physical examinations, SPT or ICTs, and environmental surveys, when available. The results of the present study indicate a predominance of IgE-mediated symptoms in patients with putative mold-induced illness. Most patients presented with symptoms of rhinitis and cough. Additional probable IgE-mediated symptoms reported by the patients were ocular pruritus, wheezing, chest tightness, and shortness of breath. These symptoms correlate with the 53% positive SPT or ICT reactions to molds, but the reactivity in 33% of the patients to nonmold environmental antigens, including trees, grasses, ragweed, mites, and house dust, confounds this correlation.

It has been established that IgE-mediated symptoms can be induced by molds in the environment.¹⁴⁻¹⁸ Burge¹⁵ reported that up to 10% of the general population and 40% of patients with asthma may experience mold allergy. In the present study, 57% of the patients with asthma and 89% of those with allergic rhinitis had skin test reactivity to molds. No other antigen group reached this level of reactivity. Based on the number of patients who experienced IgE-mediated symptoms and concurrently complained of mold toxic effects, these patients probably thought that they were exposed to toxic mold. However, our examination indicated that most of their symptoms, including cough, sneezing, ocular pruritus, nasal obstruction, postnasal drip, and fatigue, were consistent with a hypersensitivity reaction and not with a toxic or mycotoxin-mediated illness.

The results of the present study indicate that many patients exposed to mold-contaminated environments also have symptoms not suggestive of classic IgE-mediated mechanisms. Symptoms such as headache, nausea, vomiting, memory loss, shaking, abdominal pain, dysuria, epistaxis, anxiety, and weakness were present in at least 25% of the study population. Hypoxia secondary to reactive airway disease may account for headaches and anxiety, but this would suggest severe asthma. However, immune processes cannot readily explain the remaining symptoms. Potential etiologies for

Table 2. Common Presenting Symptoms of Patients With Mold Exposure*

Symptom	Children (n = 36)	Adults (n = 29)	Total (N = 65)
Rhinitis	21 (58)	19 (66)	40 (62)
Cough	17 (47)	17 (59)	34 (52)
Headache	10 (28)	12 (41)	22 (34)
Respiratory symptoms†	12 (33)	10 (34)	22 (34)
Ocular pruritus	12 (33)	7 (24)	19 (29)
CNS symptoms‡	0	16 (55)	16 (25)
Fatigue	2 (6)	13 (45)	15 (23)
Gastrointestinal symptoms§	6 (17)	5 (17)	11 (17)
Epistaxis	2 (6)	2 (7)	4 (6)
Dysuria	3 (8)	0	3 (5)

Abbreviation: CNS, central nervous system.

* Data are given as number (percent) of patients.

† Symptoms include dyspnea, wheeze, chest tightness, and shortness of breath.

‡ Symptoms include dizziness, anxiety, weakness, restless legs, memory loss, and shaking.

§ Symptoms include nausea, vomiting, and abdominal pain.

Table 3. Distribution of Fungal Species in Patients With Positive SPT or ICT Reactions

Species	Patients, No.
<i>Alternaria alternata</i>	5
<i>Acremonium strictum</i>	4
<i>Apiospora montagnei</i>	2
<i>Aspergillus fumigatus</i>	6
<i>Aspergillus niger</i>	2
<i>Aureobasidium pullulans</i>	5
<i>Botrytis cinerea</i>	2
<i>Candida albicans</i>	3
<i>Candida tropicalis</i>	3
<i>Cladosporium cladosporioides</i>	3
<i>Cladosporium fulvum</i>	5
<i>Chaetomium globosum</i>	5
<i>Chrysosporium pruinolum</i>	5
<i>Colletotrichum atramentarium</i>	2
<i>Drechslera sorokiniana</i>	2
<i>Drechslera spicifera</i>	5
<i>Epicoccum nigrum</i>	8
<i>Fusarium oxysporum</i>	6
<i>Geotrichum candidum</i>	2
<i>Gliocladium fimbriatum</i>	4
<i>Hypocrea rufa</i>	3
<i>Microsporum audouinii</i>	3
<i>Microsporum canis</i>	7
<i>Monilia sitophila</i>	5
<i>Mucor racemosus</i>	6
<i>Nigrospora oryzae</i>	7
<i>Paecilomyces variotii</i>	2
<i>Penicillium chrysogenum</i>	9
<i>Phoma destructiva</i>	5
<i>Rhizopus nigricans</i>	5
<i>Rhodotorula rubra</i>	9
<i>Saccharomyces cerevisiae</i>	6
<i>Scopulariopsis brevicaulis</i>	4
<i>Stachybotrys chartarum</i>	5
<i>Stemphylium herbarum</i>	10
<i>Streptomyces griseus*</i>	7
<i>Syncephalastrum species</i>	5
<i>Tetracoccusporium species</i>	3
<i>Trichothecium roseum</i>	2
<i>Trichophyton rubrum</i>	3

Abbreviation: ICT, intracutaneous test; SPT, skin prick test.

* *Streptomyces griseus* is a filamentous, "funguslike," aerobic actinomycete bacterium commonly classified among fungi.

these symptoms include mycotoxin-mediated reactions, direct irritation by mold spores, inflammation and irritation due to mold metabolites (including microbial VOCs), fungal cell wall components (eg, glucans), viral infections, and psychological factors. To evaluate these factors, additional studies, such as environmental microbial VOC or irritant levels and psychological assessments, would need to be performed.

Only adults in this population experienced CNS symptoms (including dizziness, memory loss, anxiety, weakness, shaking, and restless legs). Children did not exhibit or complain of

CNS symptoms, perhaps because children may not be physically or emotionally mature enough to recognize these symptoms. Furthermore, psychological components may account for many of the CNS symptoms (eg, somatization). Secondary gain, ie, financial or emotional benefits, may be the expected result of such reactions. In addition, legal and media promotion of toxic mold syndrome may have led to the dissemination of "classic symptoms" of mold toxic effects, thereby influencing patient responses during the medical interview. It is less likely that adolescents and children are affected by these factors.

Several of our patients were potentially exposed to mycotoxin-producing fungi, namely, *Aspergillus*, *Fusarium*, *Penicillium*, and *Stachybotrys*. These common indoor fungi have been well studied, and their mycotoxin production is well known.¹⁹ However, the health effects of mycotoxin exposure in individuals who live or work in mold-contaminated environments have not been solidified. Furthermore, appropriate methods for determining the ambient toxin concentrations and the effects on humans in controlled experiments have not been clarified. In our study, 5 patients with nonspecific symptoms had skin reactivities to toxin-producing molds, including *Stachybotrys*. These reactivities are likely to represent IgE-mediated rather than toxic reactions. The controversy regarding toxic mold syndrome has been focused on the potential toxic effects of *Stachybotrys*, and purposeful exposure to such mycotoxins with evaluation of symptoms induced is not yet feasible and may, in fact, not be ethical.

There is a paucity of evidence linking mycotoxin production by *Stachybotrys* species and nonspecific symptoms in exposed individuals. Previous studies^{20–22} have shown pulmonary dysfunction after exposure to *Stachybotrys* species spores. Serum and environmental biomarkers (eg, stachylysin) specific to *Stachybotrys chartarum* are currently being studied and may provide objective evidence of *S chartarum* exposure in the environment.²³ Without a reliable, proven assay to determine mycotoxin levels in environments and humans, the role of mycotoxins and their effect in humans, if any, are difficult to elucidate.

The present study was performed to evaluate a possible relationship between exposure to so-called toxic molds as expressed in the chief complaints and the multiple symptoms of the patients. This study, however, has several limitations. The chief complaint of the patients was centered on mold exposure. Patients were referred to a tertiary referral center from various parts of the United States. We were provided with environmental surveys that used different methods of data collection and that were performed by different investigators, reflecting "real-life" data that one would see in practice. This presents a challenge in data interpretation and comparison in a retrospective study. The feasibility of performing environmental surveys in homes across the country using a single surveyor would be impractical or impossible. In addition, a significant amount of recall bias was likely because all the new patients were required to fill out a questionnaire regarding previous symptoms and mold expo-

Table 4. Results of Skin Puncture/Prick or Intracutaneous Tests (SPTs or ICTs) for Molds and Other Environmental Aeroallergens

	Positive SPT or ICT results, No. (%)	Patients with asthma, No. (%)*	Patients with AR, No. (%) (n = 9)
Molds only	33/62 (53)	8/14 (57)	8/9 (89)
Env (other) only	22/55 (40)	5/11 (45)	6/8 (75)
Env + molds	18/55 (33)	4/11 (36)	6/8 (75)

Abbreviations: AR, allergic rhinitis; Env, environmental aeroallergens.

* One patient was previously diagnosed as having asthma and allergic rhinitis.

sure history. The patients may have been influenced by the media or their legal counsel.

The results of this study indicate that most patients experienced allergic reactions to the molds in their environment. There are additional limitations inherent to SPT and ICT. First, the sensitivity and specificity of SPT in diagnosing mold allergy depends on the potency and concentration of spores.¹⁷ Thus, allergic sensitization to molds is variable. Without uniform, controlled environmental surveys on all of our patients, the SPT or ICT result observed is difficult to interpret and compare among patients. Intracutaneous testing with dilutions of 1:500 may potentially be an irritant, leading to false-positive results. In addition, mold-allergic patients are usually reactive to several different species, and cross-reactivity among molds is relatively common.^{24,25} Therefore, a positive SPT or ICT reaction alone should not be used to diagnose clinical allergy. Last, we make the assumption that the common presenting symptoms of the patients referred to the Asthma and Allergy Center were IgE mediated. It is possible that these symptoms may be mediated by other mechanisms (eg, non-IgE-mediated rhinitis).

In conclusion, our primary objective was to evaluate the etiology of the presenting symptoms within the 65-patient group. We determined that 53% of the patients had positive SPT or ICT reactions to mold and that 59% reacted with concurrent potentially IgE-mediated symptoms of cough, rhinitis, ocular pruritus, and respiratory symptoms to at least 1 allergen, considering the limitations of the tests. Twenty-five percent of the patients had symptoms not compatible with IgE-mediated or other mechanisms and, therefore, are difficult to characterize. Mycotoxin-mediated mechanisms may account for these symptoms; however, currently there are no standardized, reliable urine, serum, or environmental mycotoxin detection assays. The etiology of these symptoms, therefore, remains unclear and warrants further investigation via a multidisciplinary approach. In this study, most patients were experiencing mold- or other antigen-induced allergy rather than toxic mold syndrome related to exposure to mycotoxins. Thus, our patients assumed that they were experiencing toxic mold syndrome when, in fact, most were experiencing hypersensitivity reactions to various antigens in their environment.

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