

Molds and illness

Allergy

1. Pollen and mold exposure impairs the work performance of employees with allergic rhinitis

Ann Allergy Asthma Immunol 2001 Oct;87(4):289-95 (ISSN: 1081-1206)
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BACKGROUND: Although quality of life studies suggest that allergic rhinitis has a substantial impact on work impairment, national survey estimates of the magnitude of this impairment have varied widely. Retrospective recall bias is likely to be a major cause of this variability. **OBJECTIVE:** This study used a nationally representative daily diary sample to obtain prospective data that improve on previous estimates of the work impairment because of allergic rhinitis. **METHODS:** The MacArthur Foundation National Survey of Daily Experience is a daily diary survey that included a nationally representative subsample of 739 employed people, each of whom provided daily reports on work performance for 1 randomly assigned week of the calendar year. National Allergy Bureau monitoring station data were merged with the survey data to study the association of time-space variation in pollen/mold exposure with impaired daily work quality and quantity. **RESULTS:** National Allergy Bureau pollen/mold counts are significantly related to work impairments only among respondents with self-reported allergic rhinitis. The average estimated monthly salary-equivalent work impairment costs associated with pollen/mold exposure for each allergy sufferer is between \$109 and \$156, with an annualized national projection of between \$5.4 billion and \$7.7 billion. **CONCLUSIONS:** The extent to which these costs can be recovered by increasing the proportion of allergy sufferers who are successfully treated remains unknown and can only be evaluated definitively in effectiveness trials.

2. Association of asthma symptoms and severity with indoor bioaerosols.

Allergy 2000 Aug;55(8):705-11 (ISSN: 0105-4538)
Ross MA; Curtis L; Scheff PA; Hryhorczuk DO; Ramakrishnan V; Wadden RA; Persky VW

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BACKGROUND: In this study, repeated measurements were made of levels of mold spores, bacteria, and dust-mite allergens over a 7-month period in the homes of asthmatics, and relationships with measures of asthma severity were evaluated. **METHODS:** A sample of 57 asthmatic individuals, living in 44 homes in East Moline, Illinois, and nearby communities, participated in a panel study. The homes were visited up to nine times during the study to collect air and dust samples. Asthma severity indicators were derived from questionnaire data and from the daily health records from the panel study. Associations between indoor levels of mold spores, bacteria, and dust-mite allergens were tested with several asthma severity indicators. **RESULTS:** There was evidence of associations between all asthma severity measures and levels of total and gram-negative bacteria, but mold-spore abundance was associated only with emergency room (ER) visits for asthma. No significant associations were found with house-dust-mite

allergen and any of the asthma severity indicators, but the levels of dust-mite allergen were low, with median concentrations of 0.18 microg/g dust Der f 1 and 0.19 microg/g dust Der p 1. **CONCLUSIONS:** Some evidence was found for associations of increased concentrations of gram-negative bacteria and mold spores with asthma severity, particularly with ER visits. No association was found between house-dust-mite allergen and asthma severity indicators; however, the mite-allergen levels in the study homes were generally well below the proposed threshold level of 2 microg/g dust.

3. Update on airborne mold and mold allergy

Allergy Asthma Proc 1999 Sep-Oct;20(5):289-92 (ISSN: 1088-5412)

Chapman JA

In considering the clinical aspects of fungal sensitivity, assessing exposure potential and clinical testing are essential. Valid prevalence data are difficult to secure. For ambient air, the Burkard Volumetric Spore Traps, or equal, capture spores best. For in-home analysis for fungi, the history and personal inspection of the house remains the most available method of assessment. Allergy skin test material is unavailable for most airborne fungi. Those that are available are not standardized. Yet the practicing allergist/clinical immunologist must select what fungal extract are available based on air sampling data and personal exposure of the patient. A major management approach with patients with proven sensitivity to fungal antigens and a clear correlation with clinical illness is avoidance of fungal sources. Immunotherapy should be considered when avoidance and well-tolerated pharmacotherapy are ineffective in controlling the patient's symptoms.

4. Indoor exposure to molds and allergic sensitization

Environ Health Perspect 2002 Jul;110(7):647-53 (ISSN: 0091-6765)

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Evidence that indoor dampness and mold growth are associated with respiratory health has been accumulating, but few studies have been able to examine health risks in relation to measured levels of indoor mold exposure. In particular, little is known about the contribution of indoor molds to the development of allergic sensitization. As a part of an ongoing study examining the effects of ambient air pollutants on respiratory health and atopic diseases in German school children, we examined the relation between viable mold levels indoors and allergic sensitization in 272 children. We examined whether allergic sensitization in children is associated with higher fungal spore count in settled house dust sampled from living room floors. Adjusting for age, sex, parental education, region of residency, and parental history of atopy, we found that mold spore counts for *Cladosporium* and *Aspergillus* were associated with an increased risk of allergic sensitization. Sensitized children exposed to high levels of mold spores (> 90th percentile) were more likely to suffer from symptoms of rhinoconjunctivitis. We conclude that elevated indoor concentrations of molds in wintertime might play a role in increasing the risk of developing atopic symptoms and allergic sensitization not only to molds but also to other common, inhaled allergens. These effects were strongest in the group of children who had lived in the same home since birth.

5. Mold allergy is a risk factor for persistent cold-like symptoms in children

Clin Pediatr (Phila) 1997 Dec;36(12):695-9 (ISSN: 0009-9228)
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In winter, children with mold allergy may develop persistent cold-like symptoms (PCLS) that often defy conventional therapy. To investigate the cause of PCLS, we enrolled 44 children (25 with PCLS and 19 controls) in a 2-year study to compare their clinical symptoms and the mold count in their homes. Children with PCLS had a higher percent of eosinophils in nasal smears as compared with those without PCLS (32% vs 26%). On a scale of 0 to 3, the PCLS group had higher symptom scores ($P < 0.001$ for all symptoms): bloodshot eyes (2.92 vs 0.79), mouth breathing (2.04 vs 0.68), rhinorrhea (2.48 vs 0.89), nasal voice (2.68 vs 1.00), postnasal drip (2.64 vs 0.47), and headache (2.72 vs 0.53) than the non-PCLS group. The clinical scores also correlated significantly with the mold count in the home (the r value ranged from 0.6716 to 0.7450). We conclude that management of children with PCLS should include decreasing humidity and enforcing environmental control to eradicate mold from inside the homes.

6. Increased prevalence of atopy among children exposed to mold in a school building]

Allergy 2001 Feb;56(2):175-9 (ISSN: 0105-4538)
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BACKGROUND: The purpose of this study was to assess the occurrence of immunoglobulin E sensitization to common environmental allergens (atopy) and new allergic diseases among schoolchildren after starting school in a water-damaged school building. The staff and pupils of a Finnish elementary school with visible water damage and mold complained of respiratory and skin symptoms. The school building was examined and widespread moisture damage was found. A control school with no visible water damage was also examined. No indication of exceptional microbial growth was found in the samples taken from this school. **METHODS:** History of allergic diseases and the year of diagnosis were established by a questionnaire. IgE antibodies to the common environmental allergens were determined from randomly selected groups from both schools. **RESULTS:** Elevated IgE values were significantly more common among the exposed children, as was the occurrence of new allergic diseases after the children started at the school. **CONCLUSIONS:** The odds ratios for the IgE values of the study groups indicated a possible relationship between exposure to microorganisms and IgE sensitization. Exposure to spores, toxins, and other metabolites of molds may have complex results with unknown immunogenic effects that may act as a nonspecific trigger for allergic sensitization leading to the development of atopy.

7. Mold allergy

Prim Care Pract 1997 May-Jun;1(2):226-8 Lippincotts
(ISSN: 1088-5471)

8. What makes a child allergic? Analysis of risk factors for allergic sensitization in preschool children from East and West Germany

Allergy Asthma Proc 1999 Jan-Feb;20(1):23-7 (ISSN: 1088-5412)

Schafer T; Kramer U; Dockery D; Vieluf D; Behrendt H; Ring J
Department of Dermatology and Allergy, Munich Technical University, Germany. Earlier epidemiologic studies within Germany found a higher frequency of allergic sensitization in West Germany. The reasons for that and the role of environmental factors in the process of allergic sensitization are not fully understood. This study aimed to determine the prevalence of positive skin-prick test results 5 years after unification and to investigate risk factors for allergic sensitization in preschool children. A total of 1235 children (5-6 years) from two West and five East German locations were skin-prick tested after the compulsory school entrance examination. Six common aero- (birch, grass, mugwort pollen, cat, HDM, alternaria) and two food allergens (egg, milk) were used and additional information was obtained by questionnaire. Of the tested children 23.3% exhibited at least one positive reaction. The prevalence of sensitization to the single allergens was as follows: grass (14.4%), birch (6.6%), mugwort pollen (4.5%), cat (8.5%), HDM (5.5%), alternaria (4.9%), egg (2.8%), and milk (3.9%). In the crude analysis significantly more children were sensitized in the East German city Magdeburg (40.2%) compared to the West German control region Borken (23.5%) (OR 2.20, CI 1.47-3.29). Dampness and visible molds were reported in 8.8% of all households, but significantly more often for East German apartments (10.3% versus West Germany 1.9%, OR 5.85, CI 2.55-16.53). Dampness and molds were associated with a higher frequency of sensitizations (40.6% versus 27.6% in unaffected homes). After controlling for sex, parental atopy, SES, family size, and smoking during pregnancy, this association remained statistically significant (OR 1.93, CI 1.19-3.12). With regard to single allergens, dampness and visible molds were significantly associated with sensitization to HDM (OR 3.37, CI 1.63-6.96), cat (OR 3.19, CI 1.11-5.74), and mugwort pollen (OR 2.86, CI 1.29-6.35). In addition, family size was inversely and linearly associated with the frequency of sensitization (OR for four, three, and two-person households: 1.10 (0.74-1.63), 1.57 (1.06-2.42), 2.70 (1.39-5.24), respectively, when compared to family size of five or more). Neither parental predisposition for atopic diseases nor parental education level influenced the prick test reactivity. We conclude that in addition to genetic predisposition, environmental factors like indoor climate and probably infectious stimuli (family size) play an important role in the process of allergic sensitization in children.

9. Association of sensitization to Alternaria allergens with asthma among school-age children

J Allergy Clin Immunol 1998 May;101(5):626-32 (ISSN: 0091-6749)
Perzanowski MS; Sporik R; Squillace SP; Gelber LE; Call R; Carter M; Platts-Mills TA

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BACKGROUND: Molds in the *Alternaria* genus, normally found on outdoor vegetation, produce some of the most common fungal allergens to elicit a skin test response.
OBJECTIVES: The objectives of this study were to evaluate a serum assay for IgE antibodies to *Alternaria* allergens and to establish the prevalence of sensitization to *Alternaria* allergens among children and adults enrolled in epidemiologic studies of asthma. In addition, the significance of sensitization to *Alternaria* allergens as a risk factor for asthma was compared with that of sensitization to indoor allergens or pollens.
METHODS: Using the Pharmacia Capsulated Hydrophobic Carrier Polymer (CAP) system, we have evaluated the significance of *Alternaria* allergens by using sera from several epidemiologic studies of asthma.
RESULTS: Comparisons between serum assays and skin test results suggest that this in vitro assay yields results similar to those for traditional RASTs and is as sensitive as skin prick testing. In each of the groups studied, sensitization to *Alternaria* allergens was more common among

asthmatic than control subjects, and in two studies the relationship was highly significant. *Alternaria* allergens were significantly associated with asthma in middle schools in Charlottesville, Virginia and Los Alamos, New Mexico but not in Albemarle County, Virginia. Logistic regression analysis of the results for the three schools identified an association between sensitization to *Alternaria* allergens and asthma independent of, but not as strong as, that found between sensitization to indoor allergens and asthma ($p < 0.001$). **CONCLUSIONS:** The Pharmacia CAP system is a useful tool for measuring specific IgE to *Alternaria* allergens. Although not as important as sensitization to dominant local indoor allergens, sensitization to *Alternaria* allergens appears to be a significant independent risk factor for asthma in children in some locations of the United States.

10. The relationships among environmental allergen sensitization, allergen exposure, pulmonary function, and bronchial hyperresponsiveness in the Childhood Asthma Management Program

J Allergy Clin Immunol 1999 Oct;104(4 Pt 1):775-85 (ISSN: 0091-6749)
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BACKGROUND: Sensitivity and exposure to indoor allergens constitutes a risk factor for the development and persistence of asthma in children. **OBJECTIVE:** Our purpose was to evaluate the relationship between sensitivity and exposure to inhaled allergens and lung function and bronchial responsiveness in a group of children ($n = 1041$) aged 8.9 ± 2.1 years with mild to moderate asthma enrolled in the Childhood Asthma Management Program (CAMP). **METHODS:** With use of the extensive CAMP baseline cross-sectional data on spirometry, bronchial responsiveness, allergen sensitivities, and household allergen levels, the relationship of sensitization and exposure to allergens to lung function and methacholine sensitivity was evaluated. Children who enrolled in CAMP stopped all antiasthma medication except rescue use of albuterol and prednisone for exacerbations during the 5- to 16-week screening period. During the last 2 of these weeks they underwent spirometry and methacholine challenge. Indoor allergen exposures were determined from questionnaires completed by the parent. Household levels of indoor allergens (mite, cat, dog, cockroach, mold) were determined on house dust samples. Allergen sensitivity was determined by percutaneous skin testing with a standard battery of allergens plus locally important pollen and fungal spores. Lung function and bronchial hyperresponsiveness were compared for children sensitive and not sensitive to both indoor and outdoor allergens on skin testing and, if sensitive, for exposed and not exposed to the allergens to which they were positive on skin testing. **RESULTS:** There was a strong direct correlation between increased sensitivity to inhaled methacholine and skin test sensitivity to tree, weed, *Alternaria*, cat, dog, and indoor molds. When the relationship was examined by stepwise regression, the skin test sensitivities showing the strongest associations with the concentration of methacholine that caused a 20% fall in FEV(1) were dog ($P = .003$), *Alternaria* ($P = .01$), and cat ($P = .05$). Children sensitive to any one of the aeroallergens tested were compared for the presence or absence of exposure to that allergen at the time that the methacholine challenge was performed. Those who were sensitive and exposed to weed and cat had greater methacholine sensitivity than those similarly sensitive but not exposed ($P = .003$ and $P = .02$, respectively). **CONCLUSIONS:** Sensitivity to dog or cat dander or *Alternaria* by skin testing was associated with increased bronchial responsiveness but not decreased lung function in children with mild to moderate asthma. These findings support the important role that sensitization to certain allergens plays in modulating bronchial responsiveness

11. Environmental factors as a cause for the increase in allergic disease

Ann Allergy Asthma Immunol 2001 Dec;87(6 Suppl 3):7-11 (ISSN: 1081-1206)
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LEARNING OBJECTIVES: To be able to understand the interaction among genetic factors, environmental exposure to allergens, and nonspecific adjuvant factors contributing to the increase in atopic diseases in developed countries. **DATA SOURCES:** Peer-reviewed literature identified by searching medical databases. **STUDY SELECTION:** Careful review of epidemiologic cross-sectional, sequential, and longitudinal population studies and, when appropriate, intervention studies. The criteria used to accept a study reporting environmental factors influencing the prevalence of allergic diseases were adopted from the report published by the US Department of Health and Education in 1964 (Hill AB, Principles of Medical Statistics, 9th Ed. New York: Oxford University Press, 1971, p. 323) **RESULTS:** There is ample evidence that specific environmental factors may cause sensitization and development of allergic symptoms and disease in susceptible individuals. It is unclear when and how long a sufficient exposure will result in clinical symptoms related to the immunoglobulin E-sensitizing agents. **CONCLUSIONS:** Environmental factors play an important role for the development and manifestation of allergic conditions in genetically predisposed subjects. It is well documented that increased exposure to indoor allergens and selected outdoor allergens (eg, grass pollen and molds) and smoking are important risk factors for development of asthma and allergic sensitization. The importance of other environmental factors is less clear and which environmental factors that cause the increase in prevalence of allergic disease is still unknown.

12. Environmental allergen analyses

Methods 1997 Sep;13(1):53-60 (ISSN: 1046-2023)
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Hopkins University School of Medicine, Baltimore, Maryland 21224, USA.

Environmental specimens (dust) from indoor home, school, and work-place environments can be evaluated for the content of aeroallergens produced by dust mite, cat, dog, cockroach, and molds, as a means of determining exposure risk and facilitating avoidance therapy. This article examines the variables that influence the levels of these allergens in indoor environments, methods for sampling, clinical laboratory assays used for testing, and interpretation of aeroallergen results for making decisions about remediation.

13. Childhood asthma and indoor allergens: the classroom may be a culprit

J Sch Nurs 2001 Oct;17(5):253-7 (ISSN: 1059-8405)
Epstien BL

Air Quality Sciences, Inc., Atlanta, GA, USA.
Asthma has become the most common chronic illness among children. Indoor environments appear to play a substantial role in the development of asthma. Recent studies indicate strong evidence of a causal relationship between exposure to certain indoor environmental pollutants and development and/or exacerbation of asthma in

susceptible individuals. Allergens of concern include those produced by dust mites, cockroaches, cats, dogs, and molds. It is important to better understand this relationship and take preventive and corrective steps to reduce or eliminate these sources in schools, homes, and day care centers. Measures include tracking of asthma and allergic response incidents; monitoring for the presence of allergens and molds; effective cleaning procedures; prompt repair of water leaks and/or moisture problems; control of indoor relative humidity; and proper operation of heating, ventilation, and air conditioning (HVAC) systems.

14. Microbiology on indoor air '99--what is new and interesting? An overview of selected papers presented in Edinburgh, August, 1999

Indoor Air 2000 Jun;10(2):74-80 (ISSN: 0905-6947)

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A multidisciplinary approach to microbiological implications of indoor air is fruitful for research as well as management of health and building problems. The Finnish and the Danish mold programs are examples of such productive collaborative studies. Dust samples taken from classrooms in schools where occupants complain of building-related symptoms (BRS) demonstrated an inflammatory potential in vitro, measured as a release of cytokine interleukin (IL)-8. An increase of the metabolite NO and liberation of tumor necrosis factor (TNF)-alpha and other cytokines during exposure were obtained in vivo, was presented based on these programs and on epidemiological studies on residential fungal contamination and health conducted in Canada and The Netherlands. New methods for assessing fungal exposure are PCA analysis for the toxigenic mold *Stachybotrys chartarum* and EPS-Asp/Pen for detecting of *Aspergillus* and *Penicillium* in dust. Based on a limited data set it is shown that emission rates of fungal spores are inversely proportional to relative humidity (RH), directly related to flow rate and to surface loading. Poor maintenance, risk constructions and risk materials are described in several studies as the main causes of water damage in buildings.

15. Introduction and summary: workshop on children's health and indoor mold exposure

Environ Health Perspect 1999 Jun;107 Suppl 3:465-8 (ISSN: 0091-6765)

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To evaluate the health consequences for children of indoor exposure to molds, an international workshop was organized with 15 scientists from eight countries. The participants agreed that exposure to molds may constitute a health threat to children resulting in respiratory symptoms in both the upper and lower airways, an increased incidence of infections, and skin symptoms. Allergy, either to molds or to other indoor agents, also presents a health risk. At very high exposure levels to specific molds, nose bleeding, hemoptysis, and pulmonary hemorrhage have been documented. Pediatricians and allergists need to obtain information about mold and dampness in the home environment when examining children with chronic respiratory symptoms, recurrent infections, or persistent fatigue and headache. Measurement techniques are available to determine exposure. Most important, the source of dampness must be eliminated and the indoor environment must be thoroughly cleaned of molds.

16. Effects of seasonal allergic rhinitis on selected cognitive abilities

Ann Allergy Asthma Immunol 2000 Apr;84(4):403-10 (ISSN: 1081-1206)
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BACKGROUND: Many allergy patients complain of slowed thinking, memory problems, and difficulty sustaining attention during their allergy seasons. **OBJECTIVE:** This study evaluated the effect of symptomatic allergic rhinitis on speed of cognitive processing, ability to divide and sustain attention, working memory, and recent verbal memory. **METHODS:** Symptomatic ragweed-allergic rhinitis patients and nonatopic control subjects did cognitive testing in, out of, and in ragweed seasons. **RESULTS:** Test results indicate that, during ragweed seasons, allergic patients experience subtle slowed speed of cognitive processing but not deficits in attention and recent memory. Some patients also have difficulties in working memory. **CONCLUSIONS:** These findings suggest that having allergic reactions to ragweed pollen causes significant cognitive difficulties in a subgroup of patients.

16. Decrements in vigilance and cognitive functioning associated with ragweed-induced allergic rhinitis

Ann Allergy Asthma Immunol 2002 Oct;89(4):372-80 (ISSN: 1081-1206)
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BACKGROUND AND OBJECTIVE: The adverse effects of untreated seasonal allergic rhinitis (AR) on performance in the workplace, school, and home are poorly understood. To delineate more clearly the impact and consequences of the disease on performance, the effect of symptomatic AR on vigilance and a wide range of cognitive functions was investigated. **METHODS:** A battery of automated neuropsychological tests was administered to asymptomatic adult subjects with histories of AR. Subjects were randomized to either a symptomatic or to an asymptomatic group. Subjects in the symptomatic group were exposed to ragweed pollen in a controlled exposure setting until they demonstrated predetermined severities of AR symptoms. Subjects in the asymptomatic group were not exposed to ragweed pollen in the environmental unit and retained a minimum symptom profile. The battery of cognitive measures was re-administered to both groups. **RESULTS:** AR had major adverse impacts on measures of vigilance. Further, AR adversely affected a broad range of cognitive functions. Specifically, subjects with AR symptoms demonstrated longer response times and decreased efficiency on measures of working memory, psychomotor speed, reasoning/computation, and divided attention as compared with asymptomatic subjects. **CONCLUSIONS:** In addition to decreased vigilance, AR was associated with decrements in speed and efficiency across several cognitive domains. This is similar to findings in research on medications and medical conditions that cause sedation. Findings may represent a link between AR and poor productivity/personal safety among AR sufferers. This suggests that these results have implications with regard to public health.

17. Effects of seasonal allergic rhinitis on fatigue levels and mood. [[Related Titles](#)]

Psychosom Med 2002 Jul-Aug;64(4):684-91 (ISSN: 0033-3174)
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OBJECTIVE: Many allergy patients complain of fatigue, moodiness, and dysphoria during their allergy seasons. This study evaluated the effect of symptomatic allergic rhinitis on both fatigue level and mood. **METHOD:** Symptomatic ragweed allergic rhinitis patients on no medications and healthy control subjects completed the Multi-Dimensional Fatigue Inventory and the Positive Affect-Negative Affect mood rating scales in an in-out-in ragweed season research design. **RESULTS:** During ragweed seasons, allergic patients reported higher levels of general fatigue and mental fatigue, but not physical fatigue, as well as reduced motivation. Patients described experiencing feelings of greater sadness and reduced pleasurable engagement. Increased anxiety or emotional distress was not reported. **CONCLUSIONS:** These findings suggest that having allergic reactions to ragweed pollen causes significant fatigue and mood changes in at least a subgroup of patients. Psychoneuroimmunology and medical genetics research suggests that allergic reactions engender biochemical changes that directly affect the central nervous system.

18. Health effects of indoor molds

Rev Environ Health 2001 Apr-Jun;16(2):97-103 (ISSN: 0048-7554)
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Molds grow readily indoors in the presence of dampness. Their visibility enables their effects to be investigated by means of questionnaire surveys, although these are subject to imprecision and potential bias. Exposure to airborne mold particles can be measured in various ways that also have disadvantages and limitations. Many surveys have been conducted on the health effects of molds; most have examined the association between molds and symptoms, although some studies have used lung function tests and other objective health indices. Most surveys suggest that indoor mold growth is associated with ill health, particularly of the respiratory tract. Knowing how important mold exposure really is in health terms is difficult, owing to the tendency for mold growth to be associated with other factors that are prejudicial to health.

19 "Indoor Air Pollution: An Introduction for Health Professionals"

Co-sponsored by: The American Lung Association (ALA), The Environmental Protection Agency (EPA), The Consumer Product Safety Commission (CPSC), and The American Medical Association (AMA)
U.S. Government Printing Office Publication No. 1994-523-217/81322, 1994

Excerpts

Health Problems Caused By

ANIMAL DANDER, MOLDS, DUST MITES, OTHER BIOLOGICALS

Key Signs/Symptoms

- recognized infectious disease
- exacerbation of asthma
- rhinitis

- conjunctival inflammation
- recurrent fever
- malaise
- dyspnea
- chest tightness
- cough

Diagnostic Leads

Infectious disease:

- Is the case related to the workplace, home, or other location?
(Note: It is difficult to associate a single case of any infectious disease with a specific site of exposure.)
- Does the location have a reservoir or disseminator of biologicals that may logically lead to exposure?

Hypersensitivity disease:

- Is the relative humidity in the home or workplace consistently above 50 percent?
- Are humidifiers or other water-spray systems in use? How often are they cleaned? Are they cleaned appropriately?
- Has there been flooding or leaks?
- Is there evidence of mold growth (visible growth or odors)?
- Are organic materials handled in the workplace?
- Is carpet installed on unventilated concrete (e.g., slab on grade) floors?
- Are there pets in the home?
- Are there problems with cockroaches or rodents?

Toxicosis and/or irritation:

- Is adequate outdoor air being provided?

- Is the relative humidity in the home or workplace above 50 percent or below 30 percent?
- Are humidifiers or other water-spray systems in use?
- Is there evidence of mold growth (visible growth or odors)?
- Are bacterial odors present (fishy or locker-room smells)?

Remedial Action

- Provide adequate outdoor air ventilation to dilute human source aerosols.
- Keep equipment water reservoirs clean and potable water systems adequately chlorinated, according to manufacturer instructions. Be sure there is no standing water in air conditioners. Maintain humidifiers and dehumidifiers according to manufacturer instructions.
- Repair leaks and seepage. Thoroughly clean and dry water-damaged carpets and building materials within 24 hours of damage, or consider removal and replacement.
- Keep relative humidity below 50 percent. Use exhaust fans in bathrooms and kitchens, and vent clothes dryers to outside.
- Control exposure to pets.
- Vacuum carpets and upholstered furniture regularly. *Note:* While it is important to keep an area as dust-free as possible, cleaning activities often re-suspend fine particles during and immediately after the activity. Sensitive individuals should be cautioned to avoid such exposure, and have others perform the vacuuming, or use a commercially available HEPA (High Efficiency Particulate Air) filtered vacuum.
- Cover mattresses. Wash bedding and soft toys frequently in water at a temperature above 130°F to kill dust mites.

Comment

Biological air pollutants are found to some degree in every home, school, and workplace. Sources include outdoor air and human occupants who shed viruses and bacteria, animal occupants (insects and other arthropods, mammals) that shed allergens, and indoor surfaces and water reservoirs where fungi and bacteria can grow, such as humidifiers²³. A number of factors allow biological agents to grow and be released into the

air. Especially important is high relative humidity, which encourages house dust mite populations to increase and allows fungal growth on damp surfaces. Mite and fungus contamination can be caused by flooding, continually damp carpet (which may occur when carpet is installed on poorly ventilated concrete floors), inadequate exhaust of bathrooms, or kitchen-generated moisture²⁴. Appliances such as humidifiers, dehumidifiers, air conditioners, and drip pans under cooling coils (as in refrigerators), support the growth of bacteria and fungi.

Components of mechanical heating, ventilating, and air conditioning (HVAC) systems may also serve as reservoirs or sites of microbial amplification²⁵. These include air intakes near potential sources of contamination such as standing water, organic debris or bird droppings, or integral parts of the mechanical system itself, such as various humidification systems, cooling coils, or condensate drain pans. Dust and debris may be deposited in the duct work or mixing boxes of the air handler.

Biological agents in indoor air are known to cause three types of human disease: infections, where pathogens invade human tissues; hypersensitivity diseases, where specific activation of the immune system causes disease; and toxicosis, where biologically produced chemical toxins cause direct toxic effects. In addition, exposure to conditions conducive to biological contamination (e.g., dampness, water damage) has been related to nonspecific upper and lower respiratory symptoms. Evidence is available that shows that some episodes of the group of nonspecific symptoms known as "sick building syndrome" may be related to microbial contamination in buildings²⁶.

Allergic Reactions

A major concern associated with exposure to biological pollutants is allergic reactions, which range from rhinitis, nasal congestion, conjunctival inflammation, and urticaria to asthma. Notable triggers for these diseases are allergens derived from house dust mites; other arthropods, including cockroaches; pets (cats, dogs, birds, rodents); molds; and protein-containing furnishings, including feathers, kapok, etc. In occupational settings, more unusual allergens (e.g., bacterial enzymes, algae) have caused asthma epidemics. Probably most proteins of non-human origin can cause asthma in a subset of any appropriately exposed population³⁴.

The role of mites as a source of house dust allergens has been known for 20 years^{34,35}. It is now possible to measure mite allergens in the environment and IgE antibody levels in patients using readily available techniques and standardized protocols. Experts have proposed provisional standards for levels of mite allergens in dust that lead to sensitization and

symptoms. A risk level where chronic exposure may cause sensitization is 2µg Der p1 (*Dermatophagoides pteronysinus* allergen I) per gram of dust (or 100 mites /g or 0.6 mg guanine /g of dust). A risk level for acute asthma in mite-allergic individuals is 10µg (Der p1) of the allergen per gram of dust (or 500 mites /g of dust).

Controlling house dust mite infestation includes covering mattresses, hot washing of bedding, and removing carpet from bedrooms. For mite allergic individuals, it is recommended that home relative humidities be lower than 45 percent. Mites desiccate in drier air (absolute humidities below 7 kg.). Vacuum cleaning and use of acaricides can be effective short-term remedial strategies. One such acaricide, Acarosan, is registered with EPA to treat carpets, furniture, and beds for dust mites.

Hypersensitivity Pneumonitis

Another class of hypersensitivity disease is hypersensitivity pneumonitis, which may include humidifier fever. Hypersensitivity pneumonitis, also called allergic alveo-litis, is a granulomatous interstitial lung disease caused by exposure to airborne antigens. It may affect from one to five percent or more of a specialized population exposed to appropriate antigens (e.g., farmers and farmers' lung, pigeon breeders and pigeon breeders' disease)³⁷. Continued antigen exposure may lead to end-stage pulmonary fibrosis. Hypersensitivity pneumonitis is frequently misdiagnosed as a pneumonia of infectious etiology. The prevalence of hypersensitivity pneumonitis in the general population is unknown.

Outbreaks of hypersensitivity pneumonitis in office buildings have been traced to air conditioning and humidification systems contaminated with bacteria and molds³⁸. In the home, hypersensitivity pneumonitis is often caused by contaminated humidifiers or by pigeon or pet bird antigens. The period of sensitization before a reaction occurs may be as long as months or even years. Acute symptoms, which occur four to six hours postexposure and recur on challenge with the offending agent, include cough, dyspnea, chills, myalgia, fatigue, and high fever. Nodules and nonspecific infiltrates may be noted on chest films. The white blood cell count is elevated, as is specific IgG to the offending antigen. Hypersensitivity pneumonitis generally responds to corticosteroids or cessation of exposure (either keeping symptomatic people out of contaminated environments or removing the offering agents).

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Mycotoxins

Another class of agents that may cause disease related to indoor airborne exposure is the mycotoxins. These agents are fungal metabolites that have toxic effects ranging from short-term irritation to immunosuppression and cancer. Virtually all the information related to diseases caused by mycotoxins concerns ingestion of contaminated food⁴⁰. However, mycotoxins are contained in some kinds of fungus spores, and these can enter the body through the respiratory tract. At least one case of neurotoxic symptoms possibly related to airborne mycotoxin exposure in a heavily contaminated environment has been reported⁴¹. Skin is another potential route of exposure to mycotoxins. Toxins of several fungi have caused cases of severe dermatosis. In view of the serious nature of the toxic effects reported for mycotoxins, exposure to mycotoxin-producing agents should be minimized.

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24.



Mold spores are allergens that can be found both indoors and out doors. There is no definite seasonal pattern to molds that grow indoors. However outdoor molds are seasonal, first appearing in early spring and thriving until the first frost.

Indoor molds are found in dark, warm, humid and musty environments such as damp basements, cellars, attics, bathrooms and laundry rooms. They are also found where fresh food is stored, in refrigerator drip trays, garbage pails, air conditioners and humidifiers.

Outdoor molds grow in moist shady areas. They are common in soil, decaying vegetation, compost piles, rotting wood and fallen leaves.

Preventive Strategies

- Use a dehumidifier or air conditioner to maintain relative humidity below 50% and keep temperatures cool.
- Vent bathrooms and clothes dryers to the outside.
- Check faucets, pipes and ductwork for leaks.
- When first turning on home or car air conditioners, leave the room or drive with the windows open for several minutes to allow mold spores to disperse.

- Remove decaying debris from the yard, roof and gutters.
- Avoid raking leaves, mowing lawns or working with peat, mulch, hay or dead wood. If you must do yard work, wear a mask and avoid working on hot, humid days.

25.

Pediatrics

Volume 101, Number 4

April 1998, pp 712-714

Toxic Effects of Indoor Molds (RE9736)

AMERICAN ACADEMY OF PEDIATRICS
Committee on Environmental Health

ABSTRACT. This statement describes molds, their toxic properties, and their potential for causing toxic respiratory problems in infants. Guidelines for pediatricians are given to help reduce exposures to mold in homes of infants. This is a rapidly evolving area and more research is ongoing.

ABBREVIATIONS. SIDS, sudden infant death syndrome; CDC, Centers for Disease Control and Prevention.

The growth of molds is pervasive throughout the outdoor environment. Given the proper conditions, molds may also proliferate in the indoor setting. Because Americans spend 75% to 90% of their time indoors,¹ they are exposed to molds that are growing indoors.

Molds readily enter indoor environments by circulating through doorways, windows, heating, ventilation systems, and air conditioning systems. Spores in the air also deposit on people and animals, making clothing, shoes, bags, and pets common carriers of mold into indoor environments. The most common indoor molds are *Cladosporium*, *Penicillium*, *Aspergillus*, and *Alternaria*.^{2,3}

Molds proliferate in environments that contain excessive moisture, such as from leaks in roofs, walls, plant pots, or pet urine.⁴⁻⁶ Many building materials are suitable nutrient sources for fungal growth. Cellulose substrates, including paper and paper products, cardboard, ceiling tiles, wood, and wood products, are particularly favorable for the growth of some molds. Other substrates such as dust, paints, wallpaper, insulation materials, drywall, carpet, fabric, and upholstery commonly support mold growth.³ Molds also may colonize near standing water.⁷⁻⁹

Some indoor molds have the potential to produce extremely potent toxins called mycotoxins.¹⁰⁻¹² Mycotoxins are lipid-soluble and are readily absorbed by the intestinal lining, airways, and skin.¹³ Species of mycotoxin-producing molds include *Fusarium*, *Trichoderma*, and *Stachybotrys*. In general, the presence of these molds indicates a long-standing water problem.

DIRECT TOXIC EFFECTS FROM MOLD EXPOSURE

The toxic effects from mold exposure are thought to be associated with exposure to toxins on the surface of the mold spores, not with the growth of the mold in the body. Until recently, there was only one published report in the United States linking airborne exposure to mycotoxins with health problems in humans.¹⁴ This report described upper respiratory tract irritation and rash in a family living in a Chicago home with a heavy growth of *Stachybotrys atra* (also known as *Stachybotrys chartarum*). The investigators documented that this mold was producing trichothecene mycotoxins. The symptoms disappeared when the amount of mold was substantially reduced.

More recently, molds that produce potent toxins have been associated with acute pulmonary hemorrhage among infants in Cleveland, Ohio.¹⁵ In November 1994, physicians and public health officials in Cleveland reported a cluster of eight cases of acute pulmonary hemorrhage and hemosiderosis that had occurred during January 1993 through November 1994 among infants in neighborhoods of eastern metropolitan Cleveland.¹⁶ Two additional cases were identified in December 1994. Pulmonary hemorrhage recurred in five of the discharged infants after they returned to their homes; of these infants, one died from pulmonary hemorrhage.

A case-control study comparing those 10 infants who had acute pulmonary hemorrhage and hemosiderosis with 30 age-matched control infants from the same area in Cleveland^[17] revealed that the infants with pulmonary hemorrhage were more likely to have resided in homes with major water damage from chronic plumbing leaks or flooding (95% confidence interval = 2.6 to infinity). The quantity of molds, including the toxigenic fungus *Stachybotrys atra*, was higher in the homes of infants with pulmonary hemorrhage than in those of controls. Simultaneous exposure to environmental tobacco smoke appeared to increase the risk of acute pulmonary hemorrhage among these infants.

Stachybotrys atra requires water-saturated cellulose-based materials for growth in buildings. In studies conducted in North America, it has been found in 2% to 3% of home environments sampled.⁸⁻¹⁸ Although *Stachybotrys atra* has been associated with gastrointestinal hemorrhaging in animals that had consumed moldy grain,¹⁹ the fungus previously had not been associated with disease in infants. Infants may be particularly susceptible to the effects of these inhaled mycotoxins because their lungs

are growing very rapidly. In an animal model, intranasal administration of toxic spores of *Stachybotrys atra* to mice resulted in severe interstitial inflammation with hemorrhagic exudates in the alveoli.²⁰

The county coroner re-examined all infant deaths in Cleveland during January 1993 through December 1995 to determine whether pulmonary hemosiderin-laden macrophages were present in the lung tissue. Postmortem examinations were reviewed for all 172 infants who died during that period, including 117 deaths attributed to sudden infant death syndrome (SIDS). Pathologic lung specimens were sectioned, stained with Prussian blue, and screened for the presence of hemosiderin. The presence of hemosiderin-laden macrophages in alveoli indicates alveolar bleeding at least 2 days before death.²¹

Hemosiderin-laden macrophages were abundantly present in the lung tissue of nine (5%) infants. Of these nine deaths, two resulted from homicide, and one had a recent history of child abuse. The other six deaths that were accompanied by hemosiderin-laden macrophages in the lung thus may have been misclassified as deaths from SIDS. All six infants had lived in the same limited geographic area as the previously described cases of pulmonary hemosiderosis.

The extent of this problem in other areas of the United States is still unknown. Further investigation is needed to establish causation and prevent further health effects if the findings in Cleveland are confirmed in other areas.

CONCLUSION

Very little is currently known about acute idiopathic pulmonary hemorrhage among infants. This is a newly recognized problem and knowledge is expected to be evolving rapidly. In view of the severity of the problem, environmental controls to eliminate water problems and to reduce the growth of indoor molds are wise. Until more is known about the etiology of idiopathic pulmonary hemorrhage, prudence dictates that pediatricians try to ensure that infants under 1 year of age are not exposed to chronically moldy, water-damaged environments.

Coroners and medical examiners should consider using the recently published *Guidelines for Death Scene Investigation of Sudden, Unexplained Infant Deaths*, which includes a question about dampness, visible standing water, or mold growth.

Little is known about the prevalence of toxigenic molds in homes, nor is it clear how extensive measures must be to achieve environments sufficiently free of molds to avoid disease. Bulk mold must be removed, followed by a thorough cleaning with soap and water. Caution must be used, because it is possible that homeowners could actually increase the

levels of mold spores in the air by attempting extensive clean-up efforts without guidance from a professional (a certified industrial hygienist or ventilation engineer). These specialists can be found in the yellow pages in the telephone directory under the listing for Industrial Hygiene Consultants. Additional research is needed before the most appropriate recommendations for home clean-up can be determined. Until then, interim guidelines have been formulated.

RECOMMENDATIONS

1. In areas where flooding has occurred, prompt cleaning of walls and other flood-damaged items with water mixed with chlorine bleach, diluted four parts water to one part bleach, is necessary to prevent mold growth. Never mix bleach with ammonia. Moldy items should be discarded.
2. Pediatricians should ask about mold and water damage in the home when they treat infants with idiopathic pulmonary hemorrhage. If mold is in the home, pediatricians should encourage parents to try to find and eliminate sources of moisture. Testing the environment for specific molds is usually not necessary. It appears to be important to clean up moldy conditions before the infant is discharged from the hospital to prevent recurrent pulmonary hemorrhage, although this needs further study. Interim clean-up guidelines are available through the Centers for Disease Control and Prevention (CDC), 1600 Clifton Rd, Atlanta, GA 30333.
3. Infants with idiopathic pulmonary hemorrhage must not be exposed to environments in which smoking occurs.
4. Pediatricians should report cases of idiopathic pulmonary hemorrhage and hemosiderosis to state health departments. A reporting form is available through the CDC.
5. Pediatricians should be aware that there is currently no method to test humans for toxigenic molds such as *Stachybotrys* or mycotoxins.
6. Infants who die suddenly without known cause should have an autopsy done including a Prussian blue stain of lung tissue to look for the presence of hemosiderin.

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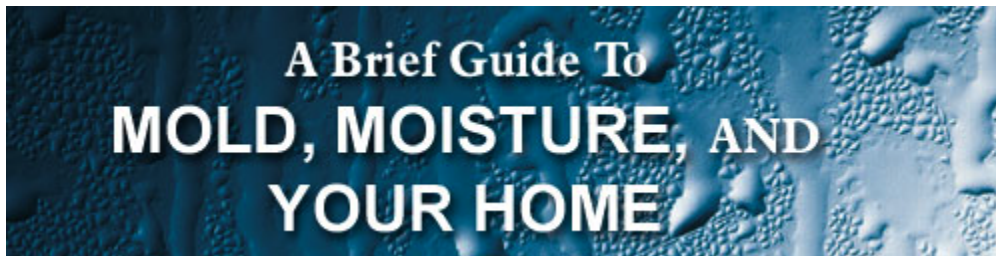
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- 26.



U.S. EPA, Office of Air and Radiation
 Indoor Environments Division (6609J)
 1200 Pennsylvania Ave., NW, Washington, DC 20460
 EPA Publication #402-K-02-003

27. TEXAS DEPARTMENT OF HEALTH

REVIEW OF PRACTICES
FOR MOLD REMEDIATION
APRIL 2002

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Ann Allergy Asthma Immunol 2002 Jul;89(1):29-33

IgE-reactive proteins from *Stachybotrys chartarum*.

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BACKGROUND: *Stachybotrys chartarum* has been associated with idiopathic pulmonary hemorrhage in infants. This is thought to be mycotoxin-related. There are increasing numbers of reports linking this fungus to the indoor environment of patients with other pulmonary problems, including allergies and asthma. **OBJECTIVE:** Given the potential significance of this fungus as a pulmonary pathogen, this work evaluates the antigenic proteins of *S. chartarum* as to their molecular size and the prevalence of immunoglobulin

(Ig)E and IgG directed against them in the general population. **METHODS:** *S. chartarum* was isolated from a local home. *S. chartarum* for extract production was grown on minimum salts and glucose. Plasma from 132 healthy individuals was evaluated for IgE and IgG directed against *S. chartarum* using direct and inhibition enzyme immunoassay. The number and molecular size of those proteins that were bound by IgE from pooled sera known to contain IgE to *S. chartarum* were determined by sodium dodecyl sulfate-polyacrylamide gel electrophoresis immunoblotting. **RESULTS:** Enzyme immunoassay indicated 65 of 132 (49.2%) sera tested contained IgG against *S. chartarum* and 13 of 139 (9.4%) sera tested contained IgE against *S. chartarum*. Pooled sera identified two IgE-binding proteins from extracts of *S. chartarum* spores and mycelia. These proteins are 34 and 52 kDa by sodium dodecyl sulfate-polyacrylamide gel electrophoresis immunoblot. **CONCLUSIONS:** We conclude sensitivity to *S. chartarum* is potentially much more widespread than previously appreciated. This fungus may impact the asthmatic and allergic population through both immunologic and toxic mechanisms. Its significance in the milieu of allergenic fungi may need to be re-evaluated.

Inflamm Res 2001 Apr;50(4):227-31

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Serum IgE specific to indoor moulds, measured by basophil histamine release, is associated with building-related symptoms in damp buildings.

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OBJECTIVE: To study the relationship between basophil histamine release (HRT) to indoor moulds, indicating specific IgE, and building-related symptoms (BRS), asthma, and hay fever in individuals working in damp and mouldy buildings. **METHODS:** A cross-sectional study was performed among 86 school staff members, who on average had worked 143 months (range: 3-396) in moist buildings with mould growth in the constructions. A questionnaire concerning mucous membrane symptoms, facial skin symptoms, central nervous system symptoms, hay fever, and asthma was fulfilled by the participants, and blood samples were taken. Eight mould species growing on building constructions were identified and cultivated to obtain allergenic materials for testing. The presence in serum of IgE specific to moulds was verified by histamine release test (HRT) based on passive sensitization of basophil leukocytes. The validity

of the method was confirmed by parallel testing of patients allergic to grass- and birch pollen and by the shift from positive to negative response after removal of serum IgE and by using sham sensitization. **RESULTS:** The prevalence of most BRS was between 32% and 62%. Positive HRT, showing serum IgE specific to one or more of the moulds, was observed in 37% of the individuals. The highest frequency of positive HRT was found to *Penicillium chrysogenum* and then to *Aspergillus* species, *Cladosporium sphaerospermum* and *Stachybotrys chartarum*. A significant association was found between most BRS and positive HRT, whereas no association was observed between positive HRT to moulds and self reported hay fever or asthma. **CONCLUSION:** Positive HRT to indoor moulds, showing the presence in serum of IgE specific to the fungi, was found to be related to BRS in individuals working in damp and mouldy buildings. Whether the association is of causal character is a question for further studies. The test may be useful in the evaluation and study of possible mould induced BRS.

Mycopathologia 2000 Jan;149(1):27-34

The time course of responses to intratracheally instilled toxic *Stachybotrys chartarum* spores in rats.

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Stachybotrys chartarum is a fungal species that can produce mycotoxins, specifically trichothecenes. Exposures in the indoor environment have reportedly induced neurogenic symptoms in adults and hemosiderosis in infants. However, little evidence has linked measured exposures to any fungal agent with any health outcome. We present here a study that focuses on quantitatively assessing the health risks from fungal toxin exposure. Male, 10 week old Charles River-Dawley rats were intratracheally instilled with approximately 9.6 million *Stachybotrys chartarum* spores in a saline suspension. The lungs were lavaged 0 h (i.e., immediately post-instillation), 6, 24 or 72 h after instillation. Biochemical indicators (albumin, myeloperoxidase, lactic dehydrogenase, hemoglobin) and leukocyte differentials in the bronchoalveolar lavage fluid and weight change were measured. We have demonstrated that a single, acute pulmonary exposure to a large quantity of *Stachybotrys chartarum* spores by intratracheal instillation causes severe injury detectable by bronchoalveolar lavage. The primary effect appears to be cytotoxicity and inflammation with hemorrhage. There is a

measurable effect as early as 6 h after instillation, which may be attributable to mycotoxins in the fungal spores. The time course of responses supports early release of some toxins, with the most severe effects occurring between 6 and 24 h following exposure. By 72 h, recovery has begun, although macrophage concentrations remained elevated.

Environ Res 2001 Mar;85(3):246-55

Preliminary description of antigenic components characteristic of *Stachybotrys chartarum*.

Raunio P, Karkkainen M, Virtanen T, Rautiainen J, Pasanen AL.

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The objective of this study was to characterize preliminarily immunogenic components characteristic of *Stachybotrys chartarum* to be used later for the development of a detection method for the fungus in environmental samples. The procedure for *S. chartarum* extract preparation was first optimized related to the age of the culture, culture type, and growth medium, and the antigenic composition of *S. chartarum* cultured in two different media was then characterized by the sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) and immunoblotting method. Cross-reactivity of *S. chartarum* antigenic components with 10 other fungal species was identified by the inhibition immunoblotting method. The 10-day-old *S. chartarum* culture extract cultured in malt extract broth revealed a wider selection of proteins and antigenic components than the 30-day-old culture extract or the culture medium extracts. When cultured in cellulose broth, *S. chartarum* produced a higher number of proteins and antigenic components than in malt extract broth. The most dominant immunogenic components of *S. chartarum* cultured in cellulose broth were those of 65, 50, 37, and 27 kDa. The components of 65 and 50 kDa proved to be the most characteristic of this fungus according to the inhibition immunoblotting analyses. Several of the *S. chartarum* components were identified as glycoproteins. Carbohydrate moieties of the *S. chartarum* components also possessed an antibody binding activity.

INTRODUCTION

The issues of whether and how to effectively remediate mold contamination have captured the attention of a large number of people, including owners and occupants of commercial and public buildings, homeowners, and environmental health professionals. Because no federal or state standards for mold remediation exist, people are often unsure how to proceed. To address these concerns, a number of governmental, professional, and industry groups have issued remediation guidance documents and recommendations. None of these guides, however, address all aspects of mold remediation. This review has been prepared to bring together information from a number of frequently consulted sources so that concerned parties can have a more comprehensive picture of current guidance on mold remediation.

The publications reviewed in this report include

Bioaerosols: Assessment and Control, American Conference of Governmental Industrial Hygienists (ACGIH)

Report of Microbial Growth Task Force, American Industrial Hygiene Association (AIHA)

Fungal Contamination in Public Buildings: A Guide to Recognition and Management, Health Canada

Guidelines on Assessment and Remediation of Fungi in Indoor Environments, New York City Department of Health (NYC DOH)

Mold Remediation in Schools and Commercial Buildings, United States Environmental Protection Agency (USEPA)

As mentioned above, these documents are widely quoted in discussions on mold remediation practices. Their inclusions in this review, however, should not necessarily be taken as an endorsement by the Texas Department of Health of all of their recommendations. By the same token, an examination of all of the existing literature on mold remediation is beyond the scope of this report. Therefore, the absence of any particular document should not be construed as a rejection of its findings or value.

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WHEN IS REMEDIATION NECESSARY?

Elimination of mold growth (i.e., “remediation”) is recommended by all of the guidance documents reviewed. Remediation includes not only removing visible mold growth but also addressing the underlying moisture problem (such as leaks or high humidity) that led to the mold growth. In some cases, remediation might include removal of mold that is “hidden” (e.g., inside a wall cavity) as well as visible mold.

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REMEDICATION METHODS

In the remediation guidelines reviewed for this document, recommended remediation criteria (such as clean-up methods, personal protective equipment, and containment required) are based upon the amount of mold present. Typically, this is expressed in terms of contaminated surface area. NYC DOH classifies mold contamination on building materials into four levels:

| Designation | Description | Area (sq ft) | Examples |
|-------------|--------------------------|---|-------------------------------------|
| Level I | Small Isolated Areas | < 10 | Ceiling tiles, small areas on walls |
| Level II | Mid-Sized Isolated Areas | 10 – 30 | Individual wallboard panels |
| Level III | Large Isolated Areas | 30 – 100 | Several wallboard panels |
| Level IV | Extensive Contamination | > 100 contiguous square feet in an area | |

NYC DOH also includes a fifth designation (Level V) for contamination in HVAC systems. Level V contains two sub-

categories: small isolated areas of contamination less than 10 sq ft (“V-a”) and areas of contamination greater than 10 sq ft (“V-b”).

USEPA and ACGIH propose three-group schemes in their remediation guidelines. USEPA classifies mold growth as Small (total surface area affected less than 10 sq ft), Medium (10 – 100 sq ft), or Large (greater than 100 sq ft OR potential for increased occupant or remediator exposure during remediation estimated to be significant). The ACGIH guidelines simply describe the contamination as Minimal, Moderate, or Extensive without giving specific areas associated with those terms. Health Canada mentions small-, medium-, and large-scale operations as being associated with areas of contamination of 0.3 sq m, 3 sq m, and 10 sq m respectively, but does not provide much detail about the associated remediation practices. HVAC (heating, ventilating, air conditioning) systems are addressed much less extensively by the other publications than by NYC DOH.

Current guidance documents agree that remediation of a mold problem includes at least two aspects, (1) identification and correction of the moisture problem(s) that caused mold growth and (2) elimination of the mold by removal of mold-contaminated materials or, under certain conditions, by cleaning the materials. Most also add that removal of dusts (including dusts generated during other remediation activities) that can contain mold fragments, spores, and toxins is an essential part of remediation. Whether mold-contaminated materials should be cleaned or discarded depends upon the nature of the materials and the extent of contamination.

Building materials can be classified as non-porous (e.g., metal, plastic, glass), semi-porous (e.g., wood, concrete), or porous (e.g., wallboard, ceiling tiles), according to their ability to absorb water. Generally, non-porous materials can be cleaned using a detergent solution and re-used. If the structural integrity of a non-porous material has been compromised, however, then NYC DOH and AIHA recommend that the material be replaced.

Re-use of a semi-porous material depends upon the extent to which fungal contamination has penetrated it. For example, surface contamination of wood can be removed by refinishing or sanding (ACGIH, AIHA), whereas semi-porous materials that

are not structurally sound or that have more than surface contamination should be discarded rather than cleaned (AIHA, NYC DOH, ACGIH). All materials of any porosity that are re-used should be dry and free of surface contamination; routine inspections of re-used materials should be conducted to assure that they remain mold-free.

As a rule, porous materials should be discarded rather than cleaned and re-used. Although NYC DOH makes allowances for re-use of contaminated porous materials, they note, “Porous materials...should be discarded if possible.” Health Canada recommends that contaminated porous materials be removed because there is no way to determine whether the cleaning has eliminated the fungal growth. ACGIH notes that porous materials not supporting active fungal growth can still be contaminated with fungal spores or particles released from other sites. USEPA does not make general remediation recommendations for the various porosities of materials. USEPA does, however, discuss four clean-up methods (wet vacuuming, damp wiping, HEPA vacuuming, and discarding contaminated materials) and makes recommendations for their use on various building and content materials under various contamination scenarios (small, medium, or large areas of contamination; see Tables 1 and 2 in the Appendix).

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ISOLATION OF WORK AREAS AND CONTAINMENT

Remediation work should be performed in such a way that the spread of contamination outside the work area is minimized. Whether containment is used, and, if so, what kind of containment is needed, depends upon the extent and type of contamination being remediated. Consultation with an environmental health professional might be necessary in order to determine properly the extent of contamination and whether containment is needed.

NYC DOH, USEPA, and ACGIH have published recommendations for containment. Increasing amounts of contamination are deemed to require progressively more containment. NYC DOH and USEPA address the issue of containment based upon the amount (in terms of surface area) of fungal contamination present. ACGIH’s recommendations are based upon the categories of Minimal, Moderate, or

Extensive visible fungal growth, with the notation that categorizing the extent of contamination involves professional judgment.

The recommendations, however, do not take into account “hidden” mold (e.g., mold present in a wall cavity). Removal of a small amount of contaminated wallboard, for example, might expose a large area of contamination that previously was not visible. In such a case, more extensive containment might be required than would be apparent based solely upon the amount of visible mold present.

In the NYC DOH guidelines, containment recommendations range from no containment necessary (Level I) to completely isolating the work area from occupied spaces, use of negative pressure with HEPA filtration, and inclusion of airlocks and a decontamination room in the containment structure (Level IV). For contamination levels I, II, and III, the use of dust suppression methods is recommended. USEPA’s containment recommendations are similar to those of the NYC DOH but go into greater detail on construction of the containment. USEPA also specifically recommends double layers of containment for the areas of heaviest contamination and that all containment areas be maintained at negative pressure.

ACGIH recommends source containment for minimally contaminated areas. Double layers of containment and negative pressure are recommended for both moderately and minimally contaminated areas. Like NYC DOH and USEPA, inclusion of airlocks and a decontamination room in the containment structure is recommended for areas of extensive contamination.

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EVACUATION OF BUILDINGS

USEPA considers protection of the health and safety of building occupants and remediators the remediation manager’s highest priority. Isolation of portions of a building undergoing remediation, described in the preceding section, can be useful in protecting both building occupants and remediation workers from the high levels of airborne fungal materials that can arise during remediation activity. In some cases, however, protecting the health of building occupants might require the evacuation of a portion or all of the building undergoing remediation.

The question of whether to vacate a building, partially or completely, during remediation is an important one. Evacuation can result in significant costs for provision of alternative living or work facilities. Evacuation can also result in lost revenue for businesses that must vacate their workspaces.

In general, current guidance documents recommend that people who experience adverse health effects associated with exposure to fungal materials should be evacuated immediately from a building undergoing remediation and remain out until the work has been completed. For other potentially affected groups, USEPA and NYC DOH offer some specific guidance for deciding whether to evacuate part or all of a building during remediation. USEPA describes at least four evacuation criteria, including:

- the size and type of the area(s) affected by mold growth;**
- the type and extent of health effects reported by building occupants;**
- the potential health risks associated with debris generated by the remediation activities; and**
- the amount of disruption likely to be caused by the remediation activities.**

USEPA also recommends that, when possible, remediation activities be carried out during “off-hours,” when the building is normally unoccupied, to minimize the effect on occupants.

NYC DOH recommends that work areas be unoccupied during remediation activities. Vacating of areas adjacent to work areas is deemed to be not necessary by NYC DOH but is recommended for potentially sensitive individuals, including infants under 12 months of age, people who have recently had surgery, immune-compromised people, and people with chronic inflammatory lung diseases. NYC DOH recommends against a building-wide evacuation unless cases of widespread fungal contamination are linked to illnesses throughout the building. NYC DOH further recommends that decisions about medical removals from an occupational setting be made by a trained occupational/environmental health practitioner, based upon the results of a clinical assessment.

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PERSONAL PROTECTIVE EQUIPMENT

The primary purpose of personal protective equipment (PPE) is to prevent the inhalation of mold and mold spores and to prevent contact of mold with the eyes or skin. The NYC DOH, USEPA, and ACGIH documents contain specific guidance on the selection of PPE for remediation of various amounts of mold contamination. Of these, the ACGIH document contains the most general guidance.

Suggested minimal PPE is presented for areas of minimal fungal growth (N-95 respirator and gloves) and areas of moderate and excessive (N-95 respirator, eye protection, and full-body covering) fungal growth. "Full-body covering" is defined as the use of full-body disposable coveralls, head cover, eye protection, gloves, and shoe coverings. The ACGIH guidance includes the notation that factors such as the nature of the contaminated material and the contaminating agent and the location of the site requiring remediation might need to be considered when selecting appropriate PPE. ACGIH also indicates that higher levels of respiratory protection might be necessary for some work and that the help of occupational and environmental health and safety professionals should be sought when selecting PPE.

NYC DOH recommends minimum PPE of respiratory protection such as an N-95 disposable respirator "in accordance with the OSHA respiratory protection standard (29 CFR 1910.134)," gloves, and eye protection be worn when remediating Level I, II, III or V-a. Level III includes the additional recommendation that a health and safety professional be consulted to provide oversight for the remediation project. For Level IV and for Level V-b, the minimum respiratory protection recommendation is full-face respirators with HEPA cartridges. At these levels, disposable protective clothing covering both head and shoes is also recommended.

USEPA, like ACGIH and NYC DOH, recommends use of an N-95 respirator, gloves, and eye protection (specifically goggles designed to prevent the entry of dust and small particles) for the smallest remediation jobs. The type of gloves used should be based upon the material being handled (e.g., whether biocides or detergents are being used). For medium-sized jobs, USEPA recommends using either limited or full PPE. Limited PPE includes a half-face or full-face air-purifying

respirator equipped with a HEPA filter cartridge and disposable paper coveralls. Full PPE includes a full-face, powered air-purifying respirator, mold-impervious head and foot coverings, and a body suit made of a breathable material, with all gaps (e.g., at wrists and ankles) sealed.

Both USEPA and NYC DOH refer to the federal Occupational Health and Safety Administration's respiratory protection standard (29 CFR 1910.134). Under 29 CFR 1910.143(d)(1)(i), private-sector employers are required to "select and provide an appropriate respirator based on the respiratory hazard(s) to which the worker is expected to be exposed and workplace and user factors that affect respirator performance and reliability."

According to AIHA, current guidance documents appear to make PPE recommendations using the assumption that remediation is always a large project and involves removal of mold-contaminated material. AIHA indicates that these documents do not appear to address situations involving very small amounts of visible mold or remediation during which contaminated materials are not removed (e.g., cleaning non-porous surfaces). AIHA suggests that in situations such as these, the use of PPE (and in particular respiratory protection) is not required. AIHA also recommends the use of the ACGIH guidelines in selecting PPE, as they include specific guidance but allow for the exercise of professional judgment in evaluating factors that vary from one situation to the next.

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USE OF BIOCIDES

Biocides are toxic chemicals or physical agents that can kill or inactivate one or more types of microorganisms (including fungi, of which molds are a subset). Examples of biocides include chlorine bleach, ozone, and ultraviolet radiation. The term "biocide" typically refers to an agent intended to kill existing microbial growth. Antifungal agents, in contrast, are chemicals that are incorporated into or applied to a material to suppress fungal growth before or as it occurs.

The literature reviewed states that the application of biocides should be at the judgment of the professional consultant addressing the specific situation. Currently available guidance documents generally advise against the routine use of biocides in remediation activities; however, no distinction is

made between the different kinds of biocides. USEPA points out that dead molds are still allergenic and, in some cases, potentially toxic. There is no evidence that the use of a biocide changes either the allergenic or the toxic properties of dead molds.

The guidance documents reason that because the purpose of remediation is the removal of mold contamination, merely killing mold that is present is not sufficient. The NYC DOH guidelines do not specifically address the issue of treating surfaces with liquid biocides (such as bleach) but recommend against the use of gaseous, vapor-phase, or aerosolized biocides. NYC DOH does note that some HVAC manufacturers recommend the use of biocides on certain parts of their systems and indicates that the manufacturers should be consulted for more information. ACGIH remarks that application of a biocide generally serves no purpose that application of a detergent or cleaning solution could not accomplish. ACGIH also indicates, however, that “if the remediation or restoration of a microbially contaminated non-porous surface or material warrants biocide use, a trained person should oversee the selection and application of a suitable biocide.”

ACGIH also discusses the use of antimicrobial (antifungal) agents, noting that they can play a role in preventing contamination on certain surfaces and finishes, especially in areas where controlling moisture is difficult. The use of antifungal agents and materials treated with them, however, does not substitute for moisture control practices and a program of routine inspection and maintenance. ACGIH also cautions that appropriate PPE should be used and precautions taken based upon the manufacturer’s directions when using antifungal agents.

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TREATMENT OF WATER-DAMAGED CONTENT ITEMS

Content items include such things as furnishings (e.g., carpet, furniture), fixtures, books, paper records, and clothing and other personal belongings. NYC DOH and Health Canada do not specifically address remediation of contaminated content items, and ACGIH does so only sparingly (e.g., “fungi growing on the surface of wood furniture may be removed by refinishing” and “carpeting and drapes that can be removed for thorough cleaning and drying may be salvageable”). AIHA

notes that content items can be damaged by mold either directly or indirectly. Direct contamination occurs when mold grows on items that have sustained water damage or have experienced extended exposure to high humidity. Indirect contamination is a result of fungal contamination that spreads from water-damaged building materials and settles on content items.

USEPA offers remediation guidance for several different classes of furnishings, including books and papers; carpet and backing; hard surface, porous flooring; and upholstered furniture and drapes (see the Appendix). The recommendations for furnishings are made along with the recommendations for building materials discussed previously, and they include the same four clean-up methods (wet vacuuming, damp wiping, HEPA vacuuming, and discarding contaminated materials). Recommendations for cleaning content items, like the recommendations for building materials, are classified according to the extent of the contamination (small, medium, or large area).

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CLEARANCE CRITERIA

At present, no state or federal agencies have established standards for “acceptable” air or surface concentrations of mold indoors. Similarly, professional organizations such as AIHA and ACGIH also have not established quantitative guidelines for air and surface mold levels in occupied spaces. Consequently, determining when a mold remediation project has been completed and judging the effectiveness of such a project must rely upon qualitative measures. The most basic of these is that people should be able to occupy or re-occupy the remediated space without health complaints or physical symptoms.

AIHA, ACGIH, and USEPA note that, for remediation to be judged successful, at least two criteria must be met: (1) the water or moisture problem that led to the mold problem must have been identified and fixed, and (2) all affected areas must have been inspected and visible mold and mold-damaged materials must have been removed. NYC DOH recommends air sampling before occupancy in cases of extensive surface contamination or HVAC system contamination (Level IV or V). If air sampling is performed, the types and concentrations of molds measured indoors should be similar to what is

measured outdoors. ACGIH adds that concentrations of biological agents in any surface samples taken should be similar to what is observed in well-maintained buildings or on construction and finishing building materials. AIHA recommends HEPA vacuuming the remediation site before occupancy, and USEPA recommends revisiting the site of remediation shortly after work is completed to ensure that there are no signs of water damage or mold growth.

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HAZARD COMMUNICATION

Information about the potential hazards associated with mold growth in a building and remediation activities should be communicated to both the workers involved in the remediation and the occupants of the building. NYC DOH recommends training building maintenance staff who will conduct remediation work on the potential health hazards of mold. This training can be conducted as part of the training needed to comply with the OSHA Hazard Communication Standard (29 CFR 1910.1200). Health Canada suggests that building maintenance personnel and maintenance staff be aware of potential problems associated with contaminated indoor air, and USEPA indicates that remediation workers, and particularly those with health-related concerns, might wish to consult with a health-care provider before working on mold remediation or investigating potentially moldy areas.

Both USEPA and NYC DOH recommend communication with building occupants throughout the remediation process. When mold contamination requiring a large-scale response is found, building occupants should be notified of that fact and given a description and timetable of the activities that will take place. The form (e.g., memos, meetings) and extent of communication will depend upon the degree of contamination and nature of the remediation work. USEPA notes that frequent and open communication maximizes the amount of time available for remediation work by addressing issues and concerns as they arise.

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HVAC (HEATING, VENTILATING, AIR CONDITIONING) SYSTEMS

Guidance on remediation of contaminated HVAC systems is less thorough than the guidance for building materials. ACGIH indicates that its recommendations on containment and PPE for building materials also can be applied to remediating surface mold growth in HVAC systems. Contaminated porous materials should be removed down to the bare metal layer and discarded appropriately. Full-scale containment is recommended by ACGIH when large areas of contaminated porous material are removed from HVAC systems, while source or local containment might be sufficient for removing small areas of contamination.

NYC DOH recommendations for remediation of HVAC systems (Level V) are divided into two scenarios, with 10 sq ft of contamination being the dividing line between them. In general, recommendations for smaller areas of contamination (Level V-a) are similar to those for Levels I-II, while guidance for larger areas (Level V-b) is similar to that given for Levels III-IV. NYC DOH recommends that the HVAC system be shut down during remediation activities. For Level V-b, air monitoring is recommended prior to re-occupancy with the HVAC system in operation. Biocide use on HVAC systems is mentioned by NYC DOH as recommended by some HVAC manufacturers. ACGIH, in contrast, considers biocide use on HVAC systems not acceptable.

USEPA recommends not running the HVAC system if mold contamination is present or suspected. Otherwise, USEPA offers little guidance for HVAC system remediation, instead referring readers to the EPA guide *Should You Have the Air Ducts in Your Home Cleaned?* ACGIH also refers to this publication. The primary focus of the EPA guide, however, as the title implies, is on general air duct cleaning and selection of a cleaning company and not on how to remediate mold contamination in HVAC systems.

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HIDDEN MOLD

Currently existing mold remediation guidelines base many of their recommendations (e.g., for appropriate PPE and containment) on the amount of visible mold that is present. In

some cases, however, indoor mold growth is not visible. Mold can grow in such “hidden” locations as the back side of wallpaper, wallboard, or dry wall; the top of ceiling tiles; under carpet or padding; in pipe chases and utility tunnels; and inside HVAC systems (e.g., drain pans in air handling units or porous duct liners). How to deal with hidden mold growth, although potentially important, receives relatively little treatment in the guidance documents.

ACGIH and NYC DOH do not address hidden mold. Health Canada describes techniques for investigating possible sources of hidden mold but does not discuss remediation. AIHA deems prudent the inclusion of hidden mold growth as an indicator for remediation while noting “the questions of whether the density and extent of fungal growth should be indicators for remediation has not been answered.” Also unanswered, according to AIHA, is whether secondary contamination (contamination resulting from active growth in another location) of hidden spaces indicates a need for remedial action.

USEPA recommends using PPE when potential sites of hidden mold contamination might be disturbed, because of the possibility of investigator exposure when hidden mold is disturbed. Consulting an experienced environmental health or remediation professional is recommended if a hidden mold problem is suspected. If hidden mold is discovered, USEPA recommends revising the remediation plan to account for the increased area affected by mold growth.

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TRAINING AND QUALIFICATIONS OF REMEDIATION WORKERS

The issue of training for and qualifications of remediation workers is discussed only briefly in the currently available guidance documents, perhaps because no national training or certification standards exist presently. Often, the references to training are general. For example, NYC DOH notes that building maintenance personnel who conduct remediation should “receive training on proper clean up methods, personal protection, and potential health hazards.” What this training would involve is not explained. NYC DOH indicates that training for building maintenance personnel can be performed as part of a program to comply with the requirements of the

OSHA hazard communication standard (29 CFR 1910.1200). The hazard communication standard, however, addresses hazardous chemicals in the workplace but does not offer guidance on the types of training needed for handling mold or other biological agents.

For areas of heavier contamination, NYC DOH recommends that personnel doing the remediation work be trained “in the handling of hazardous materials and equipped with respiratory protection in accordance with the OSHA respiratory protection standard (29 CFR 1910.134).” NYC DOH does not explain, however, what “training in the handling of hazardous materials” means. USEPA also points out that individuals wearing certain types of respiratory protection “must be trained, must have medical clearance, and must be fit-tested by a trained professional” but does not specify what type of training is required.

Health Canada offers slightly more specific guidance, noting that handling and manipulating potentially contaminated materials should be done only by individuals who have received training under the Canadian Workplace Hazardous Materials Information System (similar to the OSHA hazard communication standard, addressing primarily chemicals in the workplace) and provincial Occupational Health and Safety Acts. The training must include information on possible hazards; effective strategies to protect building occupants and remediators; and reminders to wear appropriate PPE.

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SUMMARY

The document provides a review of some of the most frequently cited publications on mold remediation. Before implementing any of the guidance described in this review, readers are advised to refer to the original source documents. In addition, individuals who are unfamiliar with or uncertain about needed remediation techniques and activities should consult with occupational and environmental health and safety professionals to determine the actions that are required in their situations.

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AIHA: *Report of Microbial Growth Task Force*. Fairfax, VA: American Industrial Hygiene Association Press, 2001. Ordering information is available at <http://www.aiha.org/product.asp?catalog%5Fname=AIHA+Marketplace+Combined&category%5Fname=&product%5Fid=458%2DEQ%2D01>

Health Canada: *Fungal Contamination in Public Buildings: A Guide to Recognition and Management*. Ottawa, Ontario: Health Canada, Environmental Health Directorate, Federal-Provincial Committee on Environmental and Occupational Health, 1995. On the Internet at http://www.hc-sc.gc.ca/ehp/ehd/catalogue/bch_pubs/fungal.pdf

New York City Department of Health: *Guidelines on Assessment and Remediation of Fungi in Indoor Environments*. New York, NY: New York City Department of Health, Bureau of Environmental & Occupational Disease Epidemiology, 2000. On the Internet at <http://www.ci.nyc.ny.us/html/doh/html/epi/moldrpt1.html>

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Radiation, Indoor Environments Division, 2001. On the Internet at

<http://www.epa.gov/iaq/molds/index.html> or
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USEPA: *Should You Have the Air Ducts in Your Home Cleaned?* (EPA Publication No. 402-K-97-002). Washington, DC: U.S. Environmental Protection Agency, Office of Air and Radiation, Indoor Environments Division, 1997. On the Internet at <http://www.epa.gov/iaq/pubs/airduct.html>

Carcinogenesis by Fungal Products

Authors:

[Garner RC](#)

Source: British Medical Bulletin, Vol. 36, No. 1, pages 47-52, 33 references, 1980/1980

Abstract:

Recent work on aflatoxins and related compounds was reviewed. Evidence indicated that, while these chemicals were potent carcinogens in the rat, they were almost without activity in the mouse and hamster. Such inconsistencies made it difficult to predict from the animal studied whether man was at risk from these chemicals in regard to the development of cancer. Human populations which were heavily exposed to aflatoxins were not usually located in areas where there were well equipped hospitals or where medical records were adequately kept to allow the conducting of an epidemiological survey, making cause and effect studies particularly difficult. The metabolism and activation of aflatoxin-B1 (1162658) was discussed in detail along with information concerning the mutagenicity of aflatoxin-B1-8,9-oxide (with test species such as Salmonella strains, Neurospora-crassa, and Drosophila-melanogaster), reaction of aflatoxin-B1-8,9-oxide (90358591) with nucleic acids, species differences in the production of aflatoxin-B1-8,9-oxide, liver slice studies, studies on aflatoxin-G1 (1165395) activation (in-vitro and in-vivo studies), chemical studies on the reactivity of the vinyl ether bond in aflatoxin-B1 and a model compound, and model compounds related to aflatoxin-B1-oxide.

Field Survey of Mycotoxin-Producing Fungi Contaminating Human Foodstuffs in Japan, with Epidemiological Background. I. Mycological and Chemical Aspects of the Detection of Mycotoxin Producers

Authors:

[Kurata H](#)
[Udagawa S](#)

[Ichinoe M](#)
[Natori S](#)
[Sakaki S](#)

Source: Symposium on Mycotoxins in Human Health, I. F. H. Purchase, Ed., The Macmillan Press LTD, London, pages 101-106, 6 references, 19711971

Abstract:

A Mycotoxin producing fungi in foods were investigated. Foodstuffs from several areas of Japan with high incidences of liver and stomach cancer were collected. Food samples were cultured and mycological examinations were performed. Mycotoxin producing fungi were widely distributed among the sampled foods, often as the dominant species. Starch foods had greater fungal contamination than fish, and small amounts of fungi were found in fermented, salted, and brined foods. The authors conclude that there is a significant mycotoxin hazard in foodstuffs in Japan, particularly as long as rice and starch are the staple foods.

Aflatoxin Exposures of Agricultural Workers

Authors:

[Burg WR](#)

Source: Institute of Environmental Health, University of Cincinnati, Cincinnati, Ohio, Terminal Progress Report, Grant R01-OH-00796, 3 pages, 5 references, 19821982

Abstract:

The exposure of farm workers to aflatoxin (1402682) was investigated. The amount of airborne aflatoxin generation by the operation of combines was examined. Sampling indicated that use of an air conditioning system in the cabs of the combines was an effective control measure. During unloading, using an cab without an air conditioning system, the levels of airborne dust reached as high as 231.1mg/m³. Aflatoxin levels varied greatly but levels as high as 195 parts per billion (ppb) were recorded. Much of the higher concentrations of aflatoxins may be in *Aspergillus-flavus* spores, which can remain airborne for some period of time. Grain elevators located near towns may be a source of hazardous emissions for the population. During harvesting the grain handlers, truckers and farmers may be exposed to highly varying amounts of aflatoxin, particularly in the southern parts of the United States. Attempts were made to develop a mathematical relationship between the level of aflatoxin in airborne dust and the concentration of aflatoxin in bulk corn. The aflatoxin entering the air appeared to be related to the method of handling and the history of the corn being processed. Significant levels of aflatoxins were found in and around large commercial grain elevators.

APPENDIX

Table 1 presents strategies to respond to water damage within 24-48 hours. These guidelines are designed to help avoid the need for remediation of mold growth by taking quick action before growth starts. If mold growth is found on the materials listed in Table 1, refer to Table 2 for guidance on remediation. Depending on the size of the area involved and resources available, professional assistance may be needed to dry an area quickly and thoroughly.

| Table 1: Water Damage - Cleanup and Mold Prevention | |
|--|---|
| Guidelines for Response to Clean Water Damage within 24-48 Hours to Prevent Mold Growth* | |
| Water-Damaged Material† | Actions |
| Books and papers | <ul style="list-style-type: none"> • • For non-valuable items, discard books and papers. • • Photocopy valuable/important items, discard originals. • • Freeze (in frost-free freezer or meat locker) or freeze-dry. |
| Carpet and backing - dry within 24-48 hours [§] | <ul style="list-style-type: none"> • • Remove water with water extraction vacuum. • • Reduce ambient humidity levels with dehumidifier. • • Accelerate drying process with fans. |
| Ceiling tiles | <ul style="list-style-type: none"> • • Discard and replace. |
| Cellulose insulation | <ul style="list-style-type: none"> • • Discard and replace. |
| Concrete or cinder block surfaces | <ul style="list-style-type: none"> • • Remove water with water extraction vacuum. • • Accelerate drying process with dehumidifiers, fans, and/or heaters. |
| Fiberglass insulation | <ul style="list-style-type: none"> • • Discard and replace. |
| Hard surface, porous flooring [§] (Linoleum, ceramic tile, vinyl) | <ul style="list-style-type: none"> • • Vacuum or damp wipe with water and mild detergent and allow to dry; scrub if necessary. • • Check to make sure |

| | |
|---|--|
| | underflooring is dry; dry underflooring if necessary. |
| Non-porous, hard surfaces (Plastics, metals) | <ul style="list-style-type: none"> • Vacuum or damp wipe with water and mild detergent and allow to dry; scrub if necessary. |
| Upholstered furniture | <ul style="list-style-type: none"> • Remove water with water extraction vacuum. • Accelerate drying process with dehumidifiers, fans, and/or heaters. • May be difficult to completely dry within 48 hours. If the piece is valuable, you may wish to consult a restoration/water damage professional who specializes in furniture. |
| Wallboard (Drywall and gypsum board) | <ul style="list-style-type: none"> • May be dried in place if there is no obvious swelling and the seams are intact. If not, remove, discard, and replace. • Ventilate the wall cavity, if possible. |
| Window drapes | <ul style="list-style-type: none"> • Follow laundering or cleaning instructions recommended by the manufacturer. |
| Wood surfaces | <ul style="list-style-type: none"> • Remove moisture immediately and use dehumidifiers, gentle heat, and fans for drying. (Use caution when applying heat to hardwood floors.) • Treated or finished wood surfaces may be cleaned with mild detergent and clean water and allowed to dry. • Wet paneling should be pried away from wall for drying. |
| <p>If mold growth has occurred or materials have been wet for more than 48 hours, consult Table 2 guidelines. Even if materials are dried within 48 hours, mold growth may have occurred. Items may be tested by professionals if there is doubt. Note that mold growth will not always occur after 48 hours; this is only a guideline. Please note that Tables 1 and 2 contain general guidelines. Their purpose is to provide basic information for remediation managers to first assess the extent of the damage and then to determine whether the remediation should be managed by in-house personnel or outside professionals. The remediation manager can then use the guidelines to help design a remediation plan or to assess a plan submitted</p> | |

by outside professionals.

[†]If a particular item(s) has high monetary or sentimental value, you may wish to consult a restoration/water damage specialist.

[§]The subfloor under the carpet or other flooring material must also be cleaned and dried. See the appropriate section of this table for recommended actions depending on the composition of the subfloor.

Source: USEPA, *Mold Remediation in Schools and Commercial Buildings*.

**Table 2: Guidelines for Remediating Building Materials
with Mold Growth Caused by Clean Water[†]**

| Material or Furnishing Affected | Cleanup Methods [†] |
|--|------------------------------|
| SMALL - Total Surface Area Affected Less Than 10 square feet (ft²) | |
| Books and papers | 3 |
| Carpet and backing | 1, 3 |
| Concrete or cinder block | 1, 3 |
| Hard surface, porous flooring (linoleum, ceramic tile, vinyl) | 1, 2, 3 |
| Non-porous, hard surfaces (plastics, metals) | 1, 2, 3 |
| Upholstered furniture & drapes | 1, 3 |
| Wallboard (drywall and gypsum board) | 3 |
| Wood surfaces | 1, 2, 3 |
| MEDIUM - Total Surface Area Affected Between 10 and 100 ft² | |
| Books and papers | 3 |
| Carpet and backing | 1,3,4 |
| Concrete or cinder block | 1,3 |
| Hard surface, porous flooring (linoleum, ceramic tile, vinyl) | 1,2,3 |
| Non-porous, hard surfaces (plastics, metals) | 1,2,3 |
| Upholstered furniture & drapes | 1,3,4 |
| Wallboard (drywall and gypsum board) | 3,4 |
| Wood surfaces | 1,2,3 |
| LARGE - Total Surface Area Affected Greater Than 100 ft² or Potential for Increased Occupant or Remediator Exposure During Remediation Estimated to be Significant | |
| Books and papers | 3 |
| Carpet and backing | 1,3,4 |

| | |
|---|---------|
| Concrete or cinder block | 1,3 |
| Hard surface, porous flooring (linoleum, ceramic tile, vinyl) | 1,2,3,4 |
| Non-porous, hard surfaces (plastics, metals) | 1,2,3 |
| Upholstered furniture & drapes | 1,3,4 |
| Wallboard (drywall and gypsum board) | 3,4 |
| Wood surfaces | 1,2,3,4 |

Table 2, continued

†Consult Table 1 if materials have been wet for less than 48 hours, and mold growth is not apparent. These guidelines are for damage caused by clean water. An experienced professional should be consulted if you and/or your remediators do not have expertise in remediating contaminated water situations.

†Select method most appropriate to situation. Since molds gradually destroy the things they grow on, if mold growth is not addressed promptly, some items may be damaged such that cleaning will not restore their original appearance. If mold growth is heavy and items are valuable or important, you may wish to consult a restoration/water damage/remediation expert. Please note that these are guidelines; other cleaning methods may be preferred by some professionals.

Cleanup Methods

- **Method 1:** Wet vacuum (in the case of porous materials, some mold spores/fragments will remain in the material but will not grow if the material is completely dried). Steam cleaning may be an alternative for carpets and some upholstered furniture.
- **Method 2:** Damp-wipe surfaces with plain water or with water and detergent solution (except wood—use wood floor cleaner); scrub as needed.
- **Method 3:** High-efficiency particulate air (HEPA) vacuum after the material has been thoroughly dried. Dispose of the contents of the HEPA vacuum in well-sealed plastic bags.
- **Method 4:** Discard—remove water-damaged materials and seal in plastic bags while inside of containment, if present. Dispose of as normal waste. HEPA vacuum area after it is dried.

Table developed from literature and remediation documents including *Bioaerosols: Assessment and Control* (American Conference of Governmental Industrial Hygienists, 1999) and *IICRC S500, Standard and Reference Guide for Professional Water Damage Restoration*, (Institute of Inspection, Cleaning and Restoration, 1999).

Source: USEPA, *Mold Remediation in Schools and Commercial Buildings*.

Evaluation of *Stachybotrys chartarum* in the house of an infant with pulmonary hemorrhage: quantitative assessment before, during, and after remediation.

Vesper S, Dearborn DG, Yike I, Allan T, Sobolewski J, Hinkley SF, Jarvis BB, Haugland RA.

**US Environmental Protection Agency, National Exposure Research Laboratory, Cincinnati, OH 45268, USA.
Vesper.Stephen@EPA.gov**

Stachybotrys chartarum is an indoor mold that has been associated with pulmonary hemorrhage cases in the Cleveland, Ohio, area. This study applied two new quantitative measurements to air samples from a home in which an infant developed PH. Quantitative polymerase chain reaction and a protein synthesis inhibition assay were used to determine the level of *S. chartarum* spores and their toxicity in air samples taken before, during, and after a remediation program was implemented to remove the fungus. Initial spore concentrations were between 0.1 and 9.3 spores/m³ of air, and the toxicity of air particulates was correspondingly low. However, the dust in the house contained between 0.4 and 2.1 x 10⁽³⁾ spores/mg (as determined by hemocytometer counts). The remediation program removed all contaminated wallboard, paneling, and carpeting in the water-damaged areas of the home. In addition, a sodium hypochlorite solution was used to spray all surfaces during remediation. Although spore counts and toxicity were high during remediation, air samples taken postremediation showed no detectable levels of *S. chartarum* or related toxicity. Nine isolates of *S. chartarum* obtained from the home were analyzed for spore toxicity, hemolytic activity, and random amplified polymorphic DNA banding patterns. None of the isolates produced highly toxic spores (>90 microg T2 toxin equivalents per gram wet weight spores) after growth for 10 and 30 days on wet wallboard, but three isolates were hemolytic consistently. DNA banding patterns suggested that

at least one of these isolates was related to isolates from homes of infants with previously investigated cases.

Asian Pac J Allergy Immunol 1995 Dec;13(2):101-5 [Related Articles](#), [Links](#)

Allergens of Bipolaris species.

Lim SH, Chew FT, Sim SM, Huang YT, Goh DY, Tan HT, Tan TK, Lee BW.

Department of Paediatrics, National University of Singapore, Singapore.

Skin prick tests done previously revealed a significantly higher percentage of sensitization to an extract of *Bipolaris* sp. among atopic individuals (34/147, 23.1%) compared to non-atopic individuals. *Bipolaris*-specific IgE levels were quantified in sera from a representative group of 38 individuals using the Fluorescence Allergosorbent Test (FAST). Result obtained by FAST were found to be comparable to the skin prick test results ($r^2 = 0.60$, $p < 0.001$ for IgE levels vs wheal sizes; $r^2 = 0.44$, $p < 0.001$ for IgE levels vs erythema sizes). Characterisation of the extract's allergenic component by sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE) showed 28 protein bands with molecular weights (MW) ranging from 11 kDa to above 100 kDa. Immunoblotting with sera of 10 *Bipolaris*-sensitive (skin prick test, 3+) individuals showed that *Bipolaris* spore extract contained at least 4 IgE binding proteins (MW 11-13 kDa, 16-17 kDa, 20-22 kDa and 36 kDa). All 10 sera reacted to the protein at MW 20-22 kDa, 2 sera with MW 11-13 kDa, 3 sera with 16-17 kDa and 6 sera with 36 kDa. This study has thus demonstrated that spores of *Bipolaris* sp. contain allergenic components which may elicit IgE-mediated reactions.

Appl Environ Microbiol 2000 Jul;66(7):2817-21

Reduction of pulmonary toxicity of *Stachybotrys chartarum* spores by methanol extraction of mycotoxins.

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The fungus *Stachybotrys chartarum* has been implicated in cases of nonspecific indoor air quality complaints in adults

and in cases of pulmonary hemorrhaging in infants. The effects that have been described have been attributed to mycotoxins. Previous dose-effect studies focused on exposure to a single mycotoxin in a solvent, a strategy which is unlikely to accurately characterize the effects of inhaled spores. In this study we examined the role of mycotoxins in the pulmonary effects caused by *S. chartarum* spores and the dose dependency of these effects. *S. chartarum* spores were extracted in methanol to reduce the mycotoxin content of the spores. Then either untreated (toxin-containing) or methanol-extracted *S. chartarum* spores were intratracheally instilled into male 10-week-old Charles River-Dawley rats. After 24 h, the lungs were lavaged, and the bronchoalveolar lavage fluid was analyzed to determine differences in lactic dehydrogenase, albumin, hemoglobin, myeloperoxidase, and leukocyte differential counts. Weight change was also monitored. Our data show that methanol extraction dramatically reduced the toxicity of *S. chartarum* spores. No statistically significant effects were observed in the bronchoalveolar lavage fluids of the animals that were treated with methanol-extracted spores at any dose. Conversely, dose-dependent effects of the toxin-containing spores were observed when we examined the lactic dehydrogenase, albumin, and hemoglobin concentrations, the polymorphonuclear leukocyte counts, and weight loss. Our findings show that a single, intense exposure to toxin-containing *S. chartarum* spores results in pulmonary inflammation and injury in a dose-dependent manner. Importantly, the effects are related to methanol-soluble toxins in the spores.

Clin Microbiol Rev 2003 Jan;16(1):144-72

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Indoor Mold, Toxigenic Fungi, and *Stachybotrys chartarum*: Infectious Disease Perspective.

Kuhn DM, Ghannoum MA.

Division of Infectious Diseases, Department of Medicine. Center for Medical Mycology, Department of Dermatology. University Hospitals of Cleveland, and Case Western Reserve University, Cleveland, Ohio 44106.

Damp buildings often have a moldy smell or obvious mold growth; some molds are human pathogens. This has caused concern

regarding health effects of moldy indoor environments and has resulted in many studies of moisture- and mold-damaged buildings. Recently, there have been reports of severe illness as a result of indoor mold exposure, particularly due to *Stachybotrys chartarum*. While many authors describe a direct relationship between fungal contamination and illness, close examination of the literature reveals a confusing picture. Here, we review the evidence regarding indoor mold exposure and mycotoxicosis, with an emphasis on *S. chartarum*. We also examine possible end-organ effects, including pulmonary, immunologic, neurologic, and oncologic disorders. We discuss the Cleveland infant idiopathic pulmonary hemorrhage reports in detail, since they provided important impetus for concerns about *Stachybotrys*. Some valid concerns exist regarding the relationship between indoor mold exposure and human disease. Review of the literature reveals certain fungus-disease associations in humans, including ergotism (*Claviceps* species), alimentary toxic aleukia (*Fusarium*), and liver disease (*Aspergillus*). While many papers suggest a similar relationship between *Stachybotrys* and human disease, the studies nearly uniformly suffer from significant methodological flaws, making their findings inconclusive. As a result, we have not found well-substantiated supportive evidence of serious illness due to *Stachybotrys* exposure in the contemporary environment. To address issues of indoor mold-related illness, there is an urgent need for studies using objective markers of illness, relevant animal models, proper epidemiologic techniques, and examination of confounding factors.

Int Arch Occup Environ Health 1996;68(4):207-18 [Related Articles](#), [Links](#)

Health and immunology study following exposure to toxigenic fungi (*Stachybotrys chartarum*) in a water-damaged office environment.

Johanning E, Biagini R, Hull D, Morey P, Jarvis B, Landsbergis P.

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There is growing concern about adverse health effects of fungal bio-aerosols on occupants of water-damaged buildings. Accidental, occupational exposure in a nonagricultural setting has not been investigated using modern immunological laboratory tests. The objective of this study was to evaluate the health status of office workers after exposure to fungal bio-aerosols, especially *Stachybotrys chartarum* (atra) (*S. chartarum*) and its toxigenic metabolites (satratoxins), and to study laboratory parameters or biomarkers related to allergic or toxic human health effects.

Exposure characterization and quantification were performed using microscopic, culture, and chemical techniques. The study population (n = 53) consisted of 39 female and 14 male employees (mean age 34.8 years) who had worked for a mean of 3.1 years at a problem office site; a control group comprised 21 persons (mean age 37.5 years) without contact with the problem office site. Health complaints were surveyed with a 187-item standardized questionnaire. A comprehensive test battery was used to study the red and white blood cell system, serum chemistry, immunology/antibodies, lymphocyte enumeration and function. Widespread fungal contamination of water-damaged, primarily cellulose material with *S. chartarum* was found. *S. chartarum* produced a macrocyclic trichothecene, satratoxin H, and spirocyclic lactones. Strong associations with exposure indicators and significant differences between employees (n = 53) and controls (n = 21) were found for lower respiratory system symptoms, dermatological symptoms, eye symptoms, constitutional symptoms, chronic fatigue symptoms and several enumeration and function laboratory tests, mainly of the white blood cell system. The proportion of mature T-lymphocyte cells (CD3%) was lower in employees than in controls, and regression analyses showed significantly lower CD3% among those reporting a history of upper respiratory infections. Specific *S. chartarum* antibody tests (IgE and IgG) showed small differences (NS). It is concluded that prolonged and intense exposure to toxigenic *S. chartarum* and other atypical fungi was associated with reported disorders of the respiratory and central nervous systems, reported disorders of the mucous membranes and a few parameters pertaining to the cellular and humoral immune system, suggesting a possible immune competency dysfunction.

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Health and immunology study following exposure to toxigenic fungi (*Stachybotrys chartarum*) in a water-damaged office environment.

Johanning E, Biagini R, Hull D, Morey P, Jarvis B, Landsbergis P.

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There is growing concern about adverse health effects of fungal bio-aerosols on occupants of water-damaged buildings. Accidental, occupational exposure in a nonagricultural setting has not been investigated using modern immunological laboratory tests. The

objective of this study was to evaluate the health status of office workers after exposure to fungal bio-aerosols, especially *Stachybotrys chartarum* (atra) (*S. chartarum*) and its toxigenic metabolites (satratoxins), and to study laboratory parameters or biomarkers related to allergic or toxic human health effects. Exposure characterization and quantification were performed using microscopic, culture, and chemical techniques. The study population (n = 53) consisted of 39 female and 14 male employees (mean age 34.8 years) who had worked for a mean of 3.1 years at a problem office site; a control group comprised 21 persons (mean age 37.5 years) without contact with the problem office site. Health complaints were surveyed with a 187-item standardized questionnaire. A comprehensive test battery was used to study the red and white blood cell system, serum chemistry, immunology/antibodies, lymphocyte enumeration and function. Widespread fungal contamination of water-damaged, primarily cellulose material with *S. chartarum* was found. *S. chartarum* produced a macrocyclic trichothecene, satratoxin H, and spirocyclic lactones. Strong associations with exposure indicators and significant differences between employees (n = 53) and controls (n = 21) were found for lower respiratory system symptoms, dermatological symptoms, eye symptoms, constitutional symptoms, chronic fatigue symptoms and several enumeration and function laboratory tests, mainly of the white blood cell system. The proportion of mature T-lymphocyte cells (CD3%) was lower in employees than in controls, and regression analyses showed significantly lower CD3% among those reporting a history of upper respiratory infections. Specific *S. chartarum* antibody tests (IgE and IgG) showed small differences (NS). It is concluded that prolonged and intense exposure to toxigenic *S. chartarum* and other atypical fungi was associated with reported disorders of the respiratory and central nervous systems, reported disorders of the mucous membranes and a few parameters pertaining to the cellular and humoral immune system, suggesting a possible immune competency dysfunction.

Toxicol Sci 2002 Feb;65(2):239-45

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Microanatomical changes in alveolar type II cells in juvenile mice intratracheally exposed to *Stachybotrys chartarum* spores and toxin.

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Stachybotrys chartarum is an important environmental fungus. We have shown recently that alveolar type II cells are sensitive to exposure to Stachybotrys chartarum spores and to the trichothecene, isosatratoxin-F, both in vitro and in vivo, in a juvenile mouse model. This sensitivity is manifest as significant changes in the composition and normal metabolic processing of pulmonary surfactant. This study evaluated the effects of a single intratracheal exposure of S. chartarum spores and toxin on ultrastructure and dimensions of alveolar type II cells from juvenile mice. This was to determine whether there are concurrent morphological and dimensional changes in the alveolar type II cell that reflect the metabolic alterations in pulmonary surfactant that we observed in the treated mice. Marked ultrastructural changes were associated with alveolar type II cells in both S. chartarum and isosatratoxin-F treated animals compared to untreated, saline, and Cladosporium cladosporioides spore treated animals. These ultrastructural changes included condensed mitochondria with separated cristae, scattered chromatin and poorly defined nucleolus, cytoplasmic rarefaction, and distended lamellar bodies with irregularly arranged lamellae. Point count stereological analysis revealed a significant increase ($p < 0.05$) in lamellar body volume density in S. chartarum and isosatratoxin-treated animals after 48 h exposure. Mitochondria volume density was significantly lower in the isosatratoxin-F (48 h exposure) and S. chartarum treated (24 and 48 h exposure) animals compared to those in the other treatment groups. These results reveal that exposure to S. chartarum spores and toxin elicit cellular responses in vivo differently from those associated with exposure to spores of a nontoxigenic mold species. They also indicate that accumulation of newly secreted pulmonary surfactant in the alveolar space of S. chartarum and isosatratoxin-F treated animals might be a consequence of cellular trauma resulting in lamellar body volume density changes leading to increased release of pulmonary surfactant into the alveolar space.

Infect Immun 2002 Apr;70(4):2065-9

Full text article at
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Stachylysin may be a cause of hemorrhaging in humans exposed to Stachybotrys chartarum.

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Ohio 45268, USA. Vesper.Stephen@EPA.gov

Stachybotrys chartarum is a toxigenic fungus that has been associated with human health concerns such as nasal bleeding in adults and pulmonary hemosiderosis (PH) in infants. Seven of eight strains of S. chartarum isolated from homes of infants with PH in Cleveland, Ohio, and the strain from the lung of an infant with PH in Texas produced stachylysin in tryptic soy broth (TSB), whereas only one out of eight strains isolated from control homes produced stachylysin. However, all strains produced stachylysin when grown on TSB with 0.7% sheep's blood. When stachylysin was injected into Lumbricus terrestris, the erythrocyte hemoglobin (absorbance peaks at 280 and 415 nm) was released, resulting in a lethal effect. These results support the hypothesis that stachylysin may be one agent responsible for hemorrhaging in humans.

J Asthma 2000 Apr;37(2):191-8

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Sick building syndrome. III. Stachybotrys chartarum.

Mahmoudi M, Gershwin ME.

**Division of Rheumatology/Allergy and Clinical Immunology,
University of California at Davis, 95616, USA.**

Increasingly, physicians are being asked to evaluate patients with putative environmentally associated illnesses. These can include a variety of problems, including infectious illnesses (Legionnaire's disease), chemical exposure in the workplace, and sick building syndromes. The latter has been an issue particularly in asthma because of the association of mold and increased bronchial responsiveness. Recently, attention has been focused on the mold Stachybotrys in human disease. Stachybotrys was first identified more than 60 years ago following an epidemic of stomatitis, rhinitis, conjunctivitis, pancytopenia, neurologic disorders, and death in horses. Since then, Stachybotrys has been identified in several outbreaks of disease in animals. It has also attracted attention as a possible agent in idiopathic pulmonary hemorrhage in infants. Stachybotrys is a relatively uncommon fungus but has been isolated from a variety of sources, including contaminated grains, tobacco, indoor air, insulator foams, and water-damaged buildings with high humidity. This fungus is particularly important because it is one of a series of fungi that produces trichothecenes mycotoxins; these mycotoxins are biologically active and can produce a variety of physiological and pathologic changes in humans and animals, including modulation of inflammation and altered alveolar surfactant

phospholipid concentrations. The presence of *Stachybotrys* in a building does not necessarily imply a cause-and-effect relationship with illness, but should alert physicians and healthcare professionals to do more vigorous environmental testing. Guidelines are presented herein for intervention measures in the maintenance of heating, ventilation, and air-conditioning systems.

Infect Immun 2001 Feb;69(2):912-6

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Initial characterization of the hemolysin stachylysin from *Stachybotrys chartarum*.

Vesper SJ, Magnuson ML, Dearborn DG, Yike I, Haugland RA.

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Stachybotrys chartarum is a toxigenic fungus that has been associated with human health concerns, including pulmonary hemorrhage and hemosiderosis. This fungus produces a hemolysin, stachylysin, which in its apparent monomeric form has a molecular mass of 11,920 Da as determined by matrix-assisted laser desorption ionization-time of flight mass spectrometry. However, it appears to form polydispersed aggregates, which confounds understanding of the actual hemolytically active form. Exhaustive dialysis or heat treatment at 60 degrees C for 30 min inactivated stachylysin. Stachylysin is composed of about 40% nonpolar amino acids and contains two cysteine residues. Purified stachylysin required more than 6 h to begin lysing sheep erythrocytes, but by 48 h, lysis was complete. Stachylysin also formed pores in sheep erythrocyte membranes.

Clin Diagn Lab Immunol 1996 Nov;3(6):645-50

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Evidence for the presence of immunoglobulin E antibodies specific to the cell wall phosphomannoproteins of *Candida albicans* in patients with allergies.

Kanbe T, Morishita M, Ito K, Tomita K, Utsunomiya K, Ishiguro A.

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To determine the major antigenic component of *Candida albicans* against immunoglobulin E (IgE) antibodies in the sera of patients with allergies who were positive for IgE antibodies to *C. albicans* crude antigen in a CAP system, phosphomannoproteins (CAMP/A or CAMP/B for serotype A or B strain, respectively) and their acid-stable portions (CAMP-S/A or CAMP-S/B) were isolated from beta-mercaptoethanol (2-ME) extracts of *C. albicans* cells of serotypes A and B, and IgE antibodies against these components were compared with those against protein complex and enolase (CAE) fractions isolated from *C. albicans* cells. The dot blot test, which was used to detect IgE antibodies to the *C. albicans* antigens, showed that IgE antibodies to the 2-ME extract and phosphomannoprotein fractions were present in the sera of 98.0% (2-ME extract), 96.8% (CAMP/A), 93.2% (CAMP-S/A), 97.2% (CAMP/B), and 81.5% (CAMP-S/B) of the patients, whereas IgE antibodies to the protein complex and CAE fractions were found in the sera of 73.6 and 48.8% of the patients, respectively. The extent of IgE binding to the 2-ME extract and phosphomannoproteins was well correlated with the fluorescence intensities estimated with the CAP system. Furthermore, the results obtained from the inhibition experiment with the CAP system indicated that the binding of IgE antibodies to *Candida* antigens is strongly inhibited by the phosphomannoprotein fraction and is an indication that the serum of the patients contained IgE antibodies specific to the cell wall phosphomannoproteins of *C. albicans*. Finally, an initial chemical analysis indicated that the epitopes for IgE antibodies on the phosphomannoproteins is a carbohydrate portion, since the ability of CAMP/A to inhibit the binding of IgE antibodies to the homologous CAMP/A was destroyed after oxidation by sodium periodate but not after digestion with proteinase K.

Phytochemistry 2000 Nov;55(6):663-73

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FULL-TEXT ARTICLE

Atranones A-G, from the toxigenic mold *Stachybotrys chartarum*.

Hinkley SF, Mazzola EP, Fettinger JC, Lam YF, Jarvis BB.

Department of Chemistry and Biochemistry, Joint Institute for Food Safety and Applied Nutrition, University of Maryland, College Park 20742, USA.

Atranones A-G have been isolated from the toxigenic fungus *Stachybotrys chartarum*. These compounds contain several unusual features including an enol-lactone as part of a 3,7-dioxabicyclo[3.3.0]octane-2-one ring system fused to an 11-membered ring. Two new dolabellane diterpenes, related in structure

to the atranones were also isolated, which suggests a diterpenoid origin for the C24 atranones.

Appl Environ Microbiol 1999 Jul;65(7):3175-81

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Hemolysis, toxicity, and randomly amplified polymorphic DNA analysis of *Stachybotrys chartarum* strains.

Vesper SJ, Dearborn DG, Yike I, Sorenson WG, Haugland RA.

National Exposure Research Laboratory, U.S. Environmental Protection Agency, Cincinnati, Ohio 45268, USA.

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Stachybotrys chartarum is an indoor air, toxigenic fungus that has been associated with a number of human and veterinary health problems. Most notable among these has been a cluster of idiopathic pulmonary hemorrhage cases that were observed in the Cleveland, Ohio, area. In this study, 16 strains of *S. chartarum* isolated from case (n = 8) or control (n = 8) homes in Cleveland and 12 non-Cleveland strains from diverse geographic locations were analyzed for hemolytic activity, conidial toxicity, and randomly amplified polymorphic DNA banding patterns. In tests for hemolytic activity, strains were grown at 23 degrees C on wet wallboard pieces for an 8-week test period. Conidia from these wallboard pieces were subcultured on sheep's blood agar once a week over this period and examined for growth and clearing of the medium at 37 or 23 degrees C. Five of the Cleveland strains (all from case homes) showed hemolytic activity at 37 degrees C throughout the 8-week test compared to 3 of the non-Cleveland strains. Five of the Cleveland strains, compared to two of the non-Cleveland strains, produced highly toxic conidia (>90 microgram of T2 toxin equivalents per g [wet weight] of conidia) after 10 and 30 days of growth on wet wallboard. Only 3 of the 28 strains examined both were consistently hemolytic and produced highly toxic conidia. Each of these strains was isolated from a house in Cleveland where an infant had idiopathic pulmonary hemorrhage.

J Occup Environ Med 1998 Mar;40(3):241-9

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- [J Occup Environ Med. 1998 Sep;40\(9\):761-4.](#)

Building-associated pulmonary disease from exposure to

Stachybotrys chartarum and Aspergillus versicolor.

Hodgson MJ, Morey P, Leung WY, Morrow L, Miller D, Jarvis BB, Robbins H, Halsey JF, Storey E.

Division of Occupational and Environmental Medicine, University of Connecticut Health Center, Farmington 06032-6210, USA.

The authors present an outbreak of disease associated with exposure to *Stachybotrys chartarum* and *Aspergillus* species. A courthouse and two associated office buildings had generated discomfort among employees for two years since initial occupancy. Multiple interventions had been unsuccessful. An initial evaluation of 14 individuals identified three with potential asthma and three with symptoms consistent with interstitial lung disease. A clinical screening protocol to identify individuals who should be removed from work identified three likely and seven possible cases of building-related asthma. Detailed environmental and engineering assessments of the building identified major problems in mechanical system design, building construction, and operational strategies leading to excess moisture and elevated relative humidities. Moisture-damaged interior surfaces in both buildings were contaminated with *S. chartarum*, *A. versicolor*, and *Penicillium* species. *Aspergillus* species, especially *A. versicolor*, at concentrations of 10(1) to 10(4)/m³ dominated the indoor air under normal operating conditions. Bulk samples also revealed large quantities of *Stachybotrys*. A questionnaire survey of the three case and two control buildings documented between three- and 15-fold increases in symptoms. A nested case-control study suggested emphysematous-like disease in individuals meeting questionnaire definitions for cases. Replication of analysis strategies used in similar previous investigations suggested an association between worsening symptoms and decreased diffusing capacity of the lung. Performance on neuropsychological measures was similar for both cases and controls, although workers with symptoms reported increased levels of current but not past psychiatric symptomatology. Chemical analyses demonstrated the presence of satratoxins G and H. Cytotoxic laboratory analyses demonstrated the presence of agents with biological effectiveness in bulk materials. No association was seen between IgE or IgG antibodies and the presence of disease. This outbreak represents a likely human response to inhaled fungal toxins in indoor environments. Moisture indoors represents a public health issue currently inadequately addressed by building, health, or housing codes.

