CHAPTER 13, SECTION 4

MOLD INVESTIGATION MEDICAL GUIDANCE

INTRODUCTION

Molds may affect human health by three mechanisms: infection (by exposure of a susceptible individual to mold spores, generally, but not always, a person who is immunocompromised), hypersensitivity (allergy to mold spores, components or toxins, including asthma and hypersensitivity pneumonitis), or direct irritation (by mycotoxins, the toxins produced by mold).

In general, results from a large indoor air study[1] found that:

- Culturable airborne fungal concentrations in indoor air are lower than those in outdoor air.
- Fungal concentrations are highest in the fall and summer and lowest in the winter and spring.
- Geographically in the continental U.S., fungal concentrations are highest in the southwest, far west, and southeast.
- The most common culturable airborne fungi, both indoors and outdoors and in all seasons and regions, were *Cladosporium*, *Penicillium*, nonsporulating fungi, and *Aspergillus*.

Considerable interest and controversy have been generated recently about searching for and identifying specific molds in buildings. The Centers for Disease Control and Prevention (CDC) currently state that determining what type of mold exists is unnecessary, and that all molds should be treated the same with respect to potential health risks and removal.[2]

Mold may proliferate almost anywhere that has too much moisture. Even if renovation is done properly, recurrence of moist conditions may lead to mold regrowth. In one study, uninstalled wallboard available from local distributors was found to contain a baseline bioburden, including *Stachybotrys chartarum*. The authors noted that sanitation and preservation treatment of the wallboard can markedly delay regrowth of certain fungi, particularly of *S. chartarum.*[3]

Mold has been considered as a causative agent of building-related illness (BRI). BRIs are caused by known pathogens, have specific symptoms, and may be serious. Specific diseases include those caused by *Legionella* species (Pontiac Fever, Legionnaire's Disease) and humidifier fever. Other airborne infectious diseases may have increased transmission when there is inadequate ventilation (e.g., Tuberculosis[4,5] Varicella,[6] and Q fever[7]).[8] Other symptoms from indoor air contamination of offices where workers shared ventilation contaminated with algal toxins (*Pfiesteria piscicida*, a dinoflagellate) are suspected to have occurred.[9]

Prudent health practice dictates limiting exposure of immunocompromised persons to excessive levels of mold spores and limiting exposure of sensitized (allergic) individuals to airborne or surface contamination of the specific mold to which the individual is sensitized.

THE PHYSICIAN'S ROLE IN THE INVESTIGATION AND RESOLUTION OF MOLD-RELATED PROBLEMS

The medical member of the indoor environmental quality (IEQ) investigative team can contribute valuable expertise in advising what organisms the industrial hygienist (IH) should sample for. If there is reason to suspect a particular species of mold - because of worker concern, fungal infection, or identification by a worker's health care provider (HCP) of allergy to a specific mold - IH can be asked to direct sampling to recover and appropriately identify that organism. Requesting IH to "sample for molds" because of non-specific symptoms in workers will generally not be helpful, since simply the presence of molds may be insignificant with respect to human health.

Communication between medical and other team members is important when trying to determine if there is an exposure pathway. For example, discovery of mold on surfaces may be incidental in a situation where airborne contamination is the problem (e.g., *Legionella*).

RETURNING WORKERS TO A BUILDING UNDER INVESTIGATION

In general, it is preferable not to keep workers out of the work area, nor to advise workers to avoid returning to a building unless: (1) a diagnosis of a building-related illness has been established, or (2) a building-related diagnosis is suspected based on symptoms, disease patterns, and findings consistent with a BRI.

If a worker is confirmed to have building-related mold allergy, the worker should not be allowed back into the building until remediation has been completed and post-remediation sampling documents reduced levels of mold. After remediation, re-exposure should be done with caution. It may be appropriate to have medical care immediately available in the case of serious allergic reactions. If remediation has been adequate, there is reason to expect the worker may successfully return to a building with few or no mold-related symptoms.[10]

The etiology of a worker's condition may be unknown, but the worker's condition is serious enough that further exposure to any potential offending agent represents an unacceptable health risk. In such cases, the prudent HCP may recommend against further exposure to a building until the medical workup is complete. However, the HCP should complete the workup thoroughly and accurately, being careful to avoid labeling the building a "health hazard" or stating that mold in the building is the etiology until after the facts have established such a link. Incorrectly identifying building mold as a source of health hazards can cause undue anxiety and loss of income among workers, decreased productivity, increased operating costs, and decreased readiness. Once a causal relationship has been established, however, relocation of affected workers to a different building may be appropriate.

A case report of office-related *Alternaria* allergy supports the following as "considerations" for concluding an association exists between IEQ-related mold exposure and illness in an occupational setting: symptoms and signs consistent with a medical diagnosis, either in vitro or in vivo evidence of exposure, environmental evidence of plausible biological exposure, and substantial improvement or resolution of the illness after appropriate building remediation.[11]

REMEDIATION

Successful remediation can result in a building that can be reoccupied without recurrent related illness, even in a subtropical climate.[12]

If sampling reveals pathogens suspected because of the symptoms or signs exhibited by building occupants, remediation effectiveness should be confirmed by clearance sampling before building reoccupation. Building processes (for example, heating, ventilation, air-conditioning and humidification systems or decorative fountains) that may be similar in other buildings may warrant preventive attention as a public health measure. Building engineers, inspectors, or public health officials may be appropriate points of contact in such situations.

INFECTIONS DUE TO MOLD

Molds are usually opportunistic pathogens, causing clinically significant infections in cases of overwhelming exposure or in individuals who are immune-compromised (i.e., debilitated by extremes of age, underlying infection, poor sanitation, inadequate nutrition, wounds), immune suppressed (chemotherapy, severe stress, pregnancy); or immune deficient (human immunodeficiency virus). A notable exception is the recent outbreak of *Cryptococcus gattii* originating in Vancouver Island, British Columbia, Canada, which infected apparently healthy adults, most likely from outdoor sources.[13] Mold infection diagnoses will be made by appropriate microbial identification studies or clinical courses (which are beyond the scope of this document).

ALLERGIES/ ALLERGENS

Respiratory or skin (allergic contact dermatitis) allergy symptoms are the most likely symptoms encountered from building-related mold allergy. Sensitivity to mold allergens is an important risk factor for adenoid hypertrophy in children with allergic rhinitis.[14] Adult-onset asthma is associated with self-reported mold exposures in the home.[15] Other organ system involvement, such as gastrointestinal hypersensitivity-related complaints, may be a clue that the offending exposure may not be indoor environment related, but rather related to an ingested allergen.

Allergens are common in most environments. Certain classes of allergens are especially pertinent to an indoor environmental quality investigation. The history given by those affected can be the most helpful information in determining the source of the problem. Buildings that have been water damaged for several days or more - whether from flood, leaking roofs or walls, broken plumbing, improperly installed or adjusted humidifiers or condensation on cold surfaces - may become culture media for any of a number of molds and fungi.

Specific IEQ-associated illnesses with an allergic (sensitization) basis include asthma, hypersensitivity pneumonitis, rhinitis or sinusitis, bronchitis or tracheitis (usually associated with sinusitis), and humidifier fever (HF). HF is thought to be allergic, as patients have shown sensitivity and symptoms with exposure to specific antigens in humidifiers.[16] HF has been associated with contamination of humidifiers by biologicals including amoeba,[17] fungi,[18, 19] *Bacillus subtilis*,[20] endotoxins, flavobacterium,[21] and *Pseudomonas* species.[22] It is also possible that not all etiologies of IEQ-related allergic complaints are biologicals, as one report noted heating, ventilation, and air conditioning system "dust and mud."[23]

Spirometry may help document involvement of the lower respiratory tract. A peak flow meter may be the simplest way to document expiratory impairment or exacerbation of asthma with building exposure.[24] A significant association was found between basophil histamine release showing serum IgE specific to one or more indoor molds, and building-related symptoms in individuals working in damp and moldy buildings.[25] Skin testing (skin prick test) may be more sensitive than blood testing (radioallergosorbent test, commonly called RAST) in detecting sensitization to molds. However, determining a specific mold to which someone is allergic in a given situation may be difficult, as sensitized individuals often react to more than one species.[26]

IRRITATION

Stachybotrys mycotoxins are biologically active,[27] and it is thought that they act as irritants. Respiratory irritation has been documented to occur in rodents exposed to *Stachybotrys*.[28] Special conditions may be necessary for mycotoxins produced by surface *Stachybotrys* in a building to reach sufficient concentrations to cause such effects, according to the results of one experimental study.[29] The controversy is noted previously as to whether documentation is sufficient that direct irritation from mycotoxins, rather than a hypersensitivity-related response to molds or mycotoxins, has occurred in humans exposed to mold in indoor air.

The primary indicator that symptoms among workers may be caused by building-related mold is that there is a temporal relationship of the symptoms to building exposure. Mold allergy may involve both IgG and IgE immunoglobulins.[30] Thus, an allergic reaction may occur immediately on entering a building, after several hours of exposure, or even 2 to 8 hours after leaving the building. A clear worker history of a temporal association of allergy symptoms with building exposure should alert the health care provider to the possibility of building-related allergy.

It is unknown how much exposure time is required before sensitivity to mold develops. As many molds are commonly found outside of the workplace, it is expected that some individuals have been sensitized prior to any occupational exposure. Further, since development of allergy to some substances may take over 30 years of exposure,[31] it is probable that in certain individuals, sensitization develops only after many years.

HEALTH CONSIDERATIONS OF SPECIFIC MOLDS

ASPERGILLUS

Aspergillus species molds are commonly found. Three types of *Aspergillus*-related lung disease are recognized: colonization of airways; allergic disease including extrinsic allergic alveolitis, asthma, allergic bronchopulmonary aspergillosis, bronchocentric granulomatosis and chronic eosinophilic pneumonia (possibly progressing to allergic granulomatosis and angiitis, also called Churg-Strauss syndrome); and invasive infections such as pseudomembranous tracheobronchitis, acute bronchopneumonia, angioinvasive aspergillosis, chronic necrotizing aspergillosis and invasive pleural disease.[32,33] It should be noted that both hypersensitivity and infection may be present simultaneously (i.e., a person with an allergic reaction to *Aspergillus* may also have an *Aspergillus* infection).[34] Inhalation of *Aspergillus* conidia or mycelium fragments may result in airway colonization, which may subsequently cause infections in susceptible hosts, and may simultaneously induce hypersensitivity (allergy).[35] A significant relationship was found between the incidence of invasive nosocomial aspergillosis and the degree of fungal contamination of air and surfaces in patient rooms in a bone marrow transplantation unit and two hematology wards.[36] As an antigen, hypersensitivity to *A. fumigatus* may cause *Aspergillus* asthma and allergic bronchopulmonary aspergillosis (ABPA).[37] Specific IgE and IgG may be detected in ABPA. Radiographic studies (x-rays) are characterized by fleeting pulmonary infiltrates that are often confused with pulmonary tuberculosis on chest x-ray, and by central bronchiectasis on chest computerized tomography (CT). Early diagnosis and therapy may alter the course of the disease and prevent the development of end-stage lung fibrosis.[38]

Aspergillus candidus, common in grain dust, has been suggested to be an etiologic factor in organic dust toxic syndrome[39] and to pose an important occupational hazard for grain handling workers through its immunomodulating properties.[40]

Aspergillus versicolor has been found in an investigation of building-related complaints, but no association was seen between IgE or IgG antibodies and the presence of disease.[41]

STACHYBOTRYS

Stachybotrys chartarum (also called *Stachybotrys atra*) has been known as an animal pathogen, and has recently attracted attention as possibly having a role in human IEQ-related disease.[42] It is a toxigenic fungus frequently found in water-damaged buildings.[43] In one study, *S. chartarum* was identified in the indoor air in 6% of the buildings studied and in the outdoor air of 1% of the buildings studied.[44] *S. chartarum* has been found to produce volatile organic compounds that are quite different from those produced by *Aspergillus*.[45]

S. chartarum produces trichothecene mycotoxins, which 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.[46] Sensitivity to *Stachybotrys* has been found to involve both immunoglobulins IgE and IgG against antigenic proteins of *S. chartarum.*[47] Effects of *S. chartarum* may be related to direct irritant as well as immunologic properties. Inhalation of *S. chartarum* extract aerosols was observed to provoke sensory irritation in the airways of both naive and immunized mice.[48] Alveolar type II cells are sensitive to exposure to *S. chartarum* spores and mycotoxin (isosatratoxin-F, a trichothecene).[49]

S. chartarum has been associated with nasal bleeding in adults. Stachylysin, a mycotoxin, may be one chemical responsible for the hemorrhagic effects.[50] Stachyrase A, a chymotrypsin-like proteinase from *S. chartarum*, has been isolated from a child with pulmonary hemorrhage.[51] A possible association between *S. chartarum* and pulmonary hemorrhage/hemosiderosis in infants has been reported, but further review of evidence by the CDC and other experts concluded that the association was unproven.[52]

Articles are not consistent as to the significance of *Stachybotrys* in relation to human health. Two reviews have found inadequate evidence to clearly establish the place of *Stachybotrys* in human disease.[53, 54]

OTHER MOLDS AND MOLD-RELATED ORGANISMS

Thermophilic *Actinomyces* and *Aspergillus fumigatus* have been suggested as possibly having a causative antigenic role in stipatosis, a hypersensitivity pneumonitis found in Mediterranean-area stucco workers exposed to those organisms in esparto grass (*Stipa tenacissima*).[55] Note that although the name *Actinomyces* suggests a fungus, actinomycosis is a bacterial infection.

Cladosporium cladosporioides was found to be the etiologic agent of hypersensitivity pneumonitis associated with a hot tub.[56] Skin sensitization to *C. cladosporioides* was the most commonly found mold skin sensitization in a small population in Toronto, Canada.[57]

Fusarium species infections in a hospital led to an investigation that identified the water distribution system of the hospital as the reservoir of *Fusarium.* Aerosolization of *Fusarium* species was documented after running the showers.[58]

IgG to *Sporobolomyces salmonicolor* was the most commonly detected anti-mold immunoglobulin associated with exposure in a Finnish military hospital building with severe, repeated, and enduring water and mold damage. Rhinitis, asthma, and alveolitis were noted among personnel reacting positively to *S. salmonicolor* provocation tests.[59]

Streptomyces albus was found to be responsible for a biopsy-proven case of hypersensitivity pneumonitis.[60]

An increased risk of developing asthma in adulthood has been found to be significantly related to IgG antibodies to *Trichoderma citrinoviride* (but not to other molds).[61]

Allergic bronchopulmonary mycosis caused by *Schizophyllum commune* in an otherwise healthy woman has been reported.[62]

Acute eosinophilic pneumonia with precipitating antibodies to *Trichosporon cutaneum*, *Trichoderma viride*, as well as *Aspergillus* species has been reported.[63] Other fungal species isolated from individuals with similar pulmonary disease include *Candida albicans*, *Penicillium*, *Geotrichum candidum*, *Stemphylium lanuginosum*, *Culvularia lunata*, and *Drechsleria hawaiensis*.[64]

For more information on IEQ medical guidance, contact Occupational Medicine at (757) 953-0769 or occmed@nehc.med.navy.mil.

REFERENCES CITED

PMID numbers are linked to article abstracts available at National Center for Biotechnology Information, U.S. National Library of Medicine, PubMed query at http://www.ncbi.nlm.nih.gov/entrez/query.fcgi

[1] Shelton BG, Kirkland KH, Flanders WD, Morris GK. Profiles of airborne fungi in buildings and outdoor environments in the United States. Appl Environ Microbiol. 2002 Apr;68(4):1743-53. PMID 11916692

[2] CDC. Questions and Answers on Stachybotrys chartarum and other molds. U.S. Department of Health and Human Services. Centers for Disease Control and Prevention. National Center for Environmental Health. June 06, 2002. Available at http://www.cdc.gov/mold/stachy.htm.

[3] Price DL, Ahearn DG. Sanitation of wallboard colonized with Stachybotrys chartarum. Curr Microbiol. 1999 Jul;39(1):21-6. PMID 10387112

[4] Burge HA. Risks associated with indoor infectious aerosols. Toxicol Ind Health. 1990 Mar;6(2):263-74. Review. PMID 2192480

[5] Nolan CM, Elarth AM, Barr H, Saeed AM, Risser DR. An outbreak of tuberculosis in a shelter for homeless men. A description of its evolution and control. Am Rev Respir Dis. 1991 Feb;143(2):257-61. PMID 1990937

[6] Tsujino G, Sako M, Takahashi M. Varicella infection in a children's hospital: prevention by vaccine and an episode of airborne transmission. Biken J 1984 Sep;27(2-3):129-32. PMID 6100050

[7] Carrieri MP, Tissot-Dupont H, Rey D, Brousse P, Renard H, Obadia Y, Raoult D. Investigation of a slaughterhouse-related outbreak of Q fever in the French Alps. Eur J Clin Microbiol Infect Dis. 2002 Jan;21(1):17-21. PMID 11913496

[8] Brundage JF, Scott RM, Lednar WM, Smith DW, Miller RN. Building-associated risk of febrile acute respiratory diseases in Army trainees. JAMA. 1988 Apr 8;259(14):2108-12. PMID 3346987

[9] Schmechel DE, Koltai DC. Potential human health effects associated with laboratory exposures to Pfiesteria piscicida. Environ Health Perspect. 2001 Oct;109 Suppl 5:775-779. PMID 11677188

[10] Jarvis JQ, Morey PR. Allergic respiratory disease and fungal remediation in a building in a subtropical climate. Appl Occup Environ Hyg. 2001 Mar;16(3):380-8. PMID 11297052

[11] Fung F, Tappen D, Wood G. Alternaria-associated asthma. Appl Occup Environ Hyg. 2000 Dec;15(12):924-7. PMID 11141604

[12] Jarvis JQ, Morey PR. Allergic respiratory disease and fungal remediation in a building in a subtropical climate. Appl Occup Environ Hyg. 2001 Mar;16(3):380-8. PMID 11297052

[13] Kidd SE, Chow Y, Mak S, Bach PJ, Chen H, Hingston AO, Kronstad JW, Bartlett KH. Characterization of Environmental Sources of the Human and Animal Pathogen, Cryptococcus gattii, in British Columbia, Canada, and Pacific Northwest USA. Appl Environ Microbiol. 2006 Dec 28. PMID 17194837

[14] Huang SW, Giannoni C. The risk of adenoid hypertrophy in children with allergic rhinitis. Ann Allergy Asthma Immunol. 2001 Oct;87(4):350-5. PMID 11686429

[15] Thorn J, Brisman J, Toren K. Adult-onset asthma is associated with self-reported mold or environmental tobacco smoke exposures in the home. Allergy. 2001 Apr;56(4):287-92. PMID 11284794

[16] Edwards JH, Cockcroft A. Inhalation challenge in humidifier fever. Clin Allergy. 1981 May;11(3):227-35. PMID 7249338

[17] Finnegan MJ, Pickering CA, Davies PS, Austwick PK, Warhurst DC. Amoebae and humidifier fever. Clin Allergy. 1987 May;17(3):235-42. PMID 3301062

[18] Mamolen M, Lewis DM, Blanchet MA, Satink FJ, Vogt RL. Investigation of an outbreak of "humidifier fever" in a print shop. Am J Ind Med. 1993 Mar;23(3):483-90. PMID 8503466

[19] Baur X, Behr J, Dewair M, Ehret W, Fruhmann G, Vogelmeier C, Weiss W, Zinkernagel V. Humidifier lung and humidifier fever. Lung. 1988;166(2):113-24. PMID 3130530

[20] Parrott WF, Blyth W. Another causal factor in the production of humidifier fever. J Soc Occup Med. 1980 Apr;30(2):63-8. PMID 6782372

[21] Rylander R, Haglind P, Lundholm M, Mattsby I, Stenqvist K. Humidifier fever and endotoxin exposure. Clin Allergy. 1978 Sep;8(5):511-6. PMID 709796

[22] Forsgren A, Persson K, Ursing J, Walder M, Borg I. Immunological aspects of humidifier fever. Eur J Clin Microbiol. 1984 Oct;3(5):411-8. PMID 6499837

[23] Lebedev SV, Aleksandrovskii VG, Chekhonin VP. Humidifier fever [Russian]. Ter Arkh. 1988;60(11):90-3. PMID 3238588

[24] National Institutes of Health. Clinical practice guidelines expert panel report 2 guidelines for the diagnosis and management of asthma. National Asthma Education and Prevention Program. National Heart, Lung, and Blood Institute. National Institutes of Health. NIH Publication No. 97-4051:July 1997. Available at http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.pdf

[25] Lander F, Meyer HW, Norn S. Serum IgE specific to indoor moulds, measured by basophil histamine release, is associated with building-related symptoms in damp buildings. Inflamm Res. 2001 Apr;50(4):227-31. PMID 11392611

[26] Mabry RL, Marple BF, Mabry CS. Mold testing by RAST and skin test methods in patients with allergic fungal sinusitis. Otolaryngol Head Neck Surg. 1999 Sep;121(3):252-4. PMID 10471866

[27] Mahmoudi M, Gershwin ME. Sick building syndrome. III. Stachybotrys chartarum. J Asthma. 2000 Apr;37(2):191-8. PMID 10805208

[28] Korpi A, Kasanen JP, Raunio P, Kosma VM, Virtanen T, Pasanen AL. Effects of aerosols from nontoxic Stachybotrys chartarum on murine airways. Inhal Toxicol. 2002 May;14(5):521-40. PMID 12028806

[29] Wilkins CK, Larsen ST, Hammer M, Poulsen OM, Wolkoff P, Nielsen GD. Respiratory effects in mice exposed to airborne emissions from Stachybotrys chartarum and implications for risk assessment. Pharmacol Toxicol. 1998 Sep;83(3):112-9. PMID 9783329

[30] Shah A, Panjabi C. Allergic bronchopulmonary aspergillosis: a review of a disease with a worldwide distribution. J Asthma. 2002 Jun;39(4):273-89. PMID 12095177

[31] Tarvainen K, Jolanki R, Estlander T. Occupational contact allergy to unsaturated polyester resin cements. Contact Dermatitis. 1993 Apr;28(4):220-4. PMID 8508632

[32] Al-Alawi A, Ryan CF, Flint JD, Muller NL. Aspergillus-related lung disease. Can Respir J. 2005 Oct;12(7):377-87. PMID 16307029

[33] Stephens M, Reynolds S, Gibbs AR, Davies B. Allergic bronchopulmonary aspergillosis progressing to allergic granulomatosis and angiitis (Churg-Strauss syndrome). Am Rev Respir Dis. 1988 May;137(5):1226-8. PMID 3195817

[34] Greene R. The pulmonary aspergilloses: three distinct entities or a spectrum of disease. Radiology. 1981 Aug;140(2):527-30. PMID 7255737

[35] Tomee JF, van der Werf TS. Pulmonary aspergillosis. Neth J Med. 2001 Nov;59(5):244-58. PMID 11705644

[36] Alberti C, Bouakline A, Ribaud P, Lacroix C, Rousselot P, Leblanc T, Derouin F. Relationship between environmental fungal contamination and the incidence of invasive aspergillosis in haematology patients. J Hosp Infect. 2001 Jul;48(3):198-206. PMID 11439007

[37] Tomee JF, van der Werf TS. Pulmonary aspergillosis. Neth J Med. 2001 Nov;59(5):244-58. PMID 11705644

[38] Shah A, Panjabi C. Allergic bronchopulmonary aspergillosis: a review of a disease with a worldwide distribution. J Asthma. 2002 Jun;39(4):273-89. PMID 12095177

[39] Krysinska-Traczyk E. Microflora of the farming work environment as an occupational risk factor [Polish]. Med Pr. 2000;51(4):351-5. PMID 11059408

[40] Krysinska-Traczyk E, Dutkiewicz J. Aspergillus candidus: a respiratory hazard associated with grain dust. Ann Agric Environ Med. 2000;7(2):101-9. PMID 11153039

[41] Hodgson MJ, Morey P, Leung WY, Morrow L, Miller D, Jarvis BB, Robbins H, Halsey JF, Storey E. Building-associated pulmonary disease from exposure to Stachybotrys chartarum and Aspergillus versicolor. J Occup Environ Med. 1998 Mar;40(3):241-9. PMID 9531095

[42] Mahmoudi M, Gershwin ME. Sick building syndrome. III. Stachybotrys chartarum. J Asthma. 2000 Apr;37(2):191-8. PMID 10805208

[43] Gao P, Martin J. Volatile metabolites produced by three strains of Stachybotrys chartarum cultivated on rice and gypsum board. Appl Occup Environ Hyg. 2002 Jun;17(6):430-6. PMID 12049433

[44] Shelton BG, Kirkland KH, Flanders WD, Morris GK. Profiles of airborne fungi in buildings and outdoor environments in the United States. Appl Environ Microbiol. 2002 Apr;68(4):1743-53. PMID 11916692

[45] Gao P, Martin J. Volatile metabolites produced by three strains of Stachybotrys chartarum cultivated on rice and gypsum board. Appl Occup Environ Hyg. 2002 Jun;17(6):430-6. PMID 12049433

[46] Mahmoudi M, Gershwin ME. Sick building syndrome. III. Stachybotrys chartarum. J Asthma. 2000 Apr;37(2):191-8. PMID 10805208

[47] Barnes C, Buckley S, Pacheco F, Portnoy J. IgE-reactive proteins from Stachybotrys chartarum. Ann Allergy Asthma Immunol. 2002 Jul;89(1):29-33. PMID 12141716

[48] Korpi A, Kasanen JP, Raunio P, Kosma VM, Virtanen T, Pasanen AL. Effects of aerosols from nontoxic Stachybotrys chartarum on murine airways. Inhal Toxicol. 2002 May;14(5):521-40. PMID 12028806

[49] Rand TG, Mahoney M, White K, Oulton M. Microanatomical changes in alveolar type II cells in juvenile mice intratracheally exposed to Stachybotrys chartarum spores and toxin.Toxicol Sci. 2002 Feb;65(2):239-45. PMID 11812928

[50] Vesper SJ, Vesper MJ. Stachylysin may be a cause of hemorrhaging in humans exposed to Stachybotrys chartarum. Infect Immun. 2002 Apr;70(4):2065-9. PMID 11895972

[51] Kordula T, Banbula A, Macomson J, Travis J. Isolation and properties of stachyrase A, a chymotrypsin-like serine proteinase from Stachybotrys chartarum. Infect Immun. 2002 Jan;70(1):419-21. PMID 11748212

[52] CDC. Update: Pulmonary hemorrhage/hemosiderosis among infants--Cleveland, Ohio, 1993-1996. MMWR Morb Mortal Wkly Rep. 2000 Mar 10;49(9):180-4. PMID 11795499

[53] Terr AI. Stachybotrys: relevance to human disease. Ann Allergy Asthma Immunol. 2001 Dec;87(6 Suppl 3):57-63. PMID 11770686

[54] Page EH, Trout DB. The role of Stachybotrys mycotoxins in building-related illness. AIHAJ. 2001 Sep-Oct;62(5):644-8. PMID 11669391

[55] Hinojosa M, Fraj J, De la Hoz B, Alcazar R, Sueiro A. Hypersensitivity pneumonitis in workers exposed to esparto grass (Stipa tenacissima) fibers. J Allergy Clin Immunol. 1996 Nov;98(5 Pt 1):985-91. PMID 8939163

[56] Jacobs RL, Thorner RE, Holcomb JR, Schwietz LA, Jacobs FO. Hypersensitivity pneumonitis caused by Cladosporium in an enclosed hot-tub area. Ann Intern Med. 1986 Aug;105(2):204-6. PMID 3729202

[57] Tarlo SM, Fradkin A, Tobin RS. Skin testing with extracts of fungal species derived from the homes of allergy clinic patients in Toronto, Canada. Clin Allergy. 1988 Jan;18(1):45-52. PMID 3349592

[58] Anaissie EJ, Kuchar RT, Rex JH, Francesconi A, Kasai M, Muller FM, Lozano-Chiu M, Summerbell RC, Dignani MC, Chanock SJ, Walsh TJ. Fusariosis associated with pathogenic fusarium species colonization of a hospital water system: a new paradigm for the epidemiology of opportunistic mold infections. Clin Infect Dis. 2001 Dec 1;33(11):1871-8. PMID 11692299

[59] Seuri M, Husman K, Kinnunen H, Reiman M, Kreus R, Kuronen P, Lehtomaki K, Paananen M. An outbreak of respiratory diseases among workers at a water-damaged building--a case report. Indoor Air. 2000 Sep;10(3):138-45. PMID 10979195

[60] Kagen SL, Fink JN, Schlueter DP, Kurup VP, Fruchtman RB. Streptomyces albus: a new cause of hypersensitivity pneumonitis. J Allergy Clin Immunol. 1981 Oct;68(4):295-9. PMID 6793652

[61] Jaakkola MS, Laitinen S, Piipari R, Uitti J, Nordman H, Haapala AM, Jaakkola JJ. Immunoglobulin G antibodies against indoor dampness-related microbes and adult-onset asthma: a population-based incident case-control study. Clin Exp Immunol. 2002 Jul;129(1):107-12. PMID 12100029

[62] Kamei K, Unno H, Nagao K, Kuriyama T, Nishimura K, Miyaji M. Allergic bronchopulmonary mycosis caused by the basidiomycetous fungus Schizophyllum commune. Clin Infect Dis. 1994 Mar;18(3):305-9. PMID 8011808

[63] Mouri M, Nambu Y, Horii H, Kobayashi Y, Yamanouchi K, Sakurai S, Toga H, Ohya N. Case report and review of literature on seasonal distribution and pathogenesis of acute eosinophilic pneumonia in Japan. [Article in Japanese]. Nihon Kyobu Shikkan Gakkai Zasshi. 1993 Dec;31(12):1578-84. PMID 8121096

[64] Lahoute C, Tonnel AB, Fournier E, Ramon P, Voisin C. Bronchopulmonary pathology with hypereosinophilia of fungal origin (excluding allergic bronchopulmonary aspergillosis). [Article in French]. Poumon Coeur. 1983;39(2):87-93. PMID 6878115