

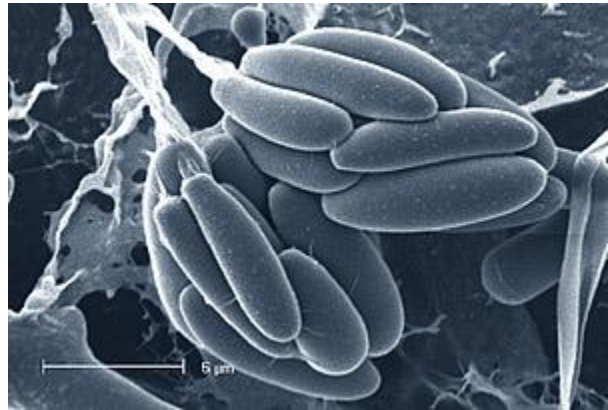
Mold & Indoor Air Quality

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Introduction

While occupational exposure to airborne pollutants such as asbestos and coal dust is known to cause lung cancer/mesothelioma and pneumoconiosis (black lung disease), consequences of exposure to air contaminants, especially bioaerosols, in homes and non-industrial work sites such as office buildings are not yet fully understood. In the 1970's and 1980's microbial contamination was identified as the primary cause for poor air quality in only 5% of more than 500 indoor air quality (IAQ) investigations conducted by National Institute for Occupational Safety and Health (NIOSH); while the remaining 95% resulted from inadequate ventilation, entrainment of outdoor air contaminants, contaminants in building fabric and unknown sources (NIOSH, 1989). However, in the last 10 years, microorganisms were the primary source of indoor air contamination in as many as 35-50% of IAQ cases (Lewis, 1994). This change has been attributed at least partially to a paradigm shift from chemical contaminant-based investigations to an interdisciplinary approach combining evaluation of physical, chemical and microbiological constituents of indoor air environments. This report specifically focuses on fungal contamination in office and home environments.



Molds in Indoor Air

Fungi are ubiquitous organisms that make up approximately 25% of earth's biomass. They can be subdivided somewhat artificially by gross morphology into yeasts, mushrooms and **molds** - the fungi of most importance for indoor air. Molds are very adaptable and can colonize dead and decaying organic matter (e.g. textiles, leather, wood, paper) and even damp, inorganic material (e.g. glass, painted surfaces, bare concrete) if organic nutrients such as dust or soil particles are available. Because various genera grow and reproduce at different substrate water concentrations and temperatures, molds occur in a wide range of habitats.

Constituents of indoor air are determined by both outdoor and indoor sources (Table 1). Likewise mold types and concentrations indoors are primarily a function of outdoor fungi and substrate water (related to indoor humidity level). Higher concentrations of outdoor molds and other fungi occur where trees, shrubs and landscape irrigation occur close to exterior building walls. (While most indoor molds originate from exterior sources, some species of *Aspergillus* and *Penicillium* can grow and reproduce effectively indoors and

are commonly found in air samples of normal, "dry" buildings.)

Molds are composed of linear chains of cells (hyphae) that branch and intertwine to form the fungus body (mycelium). All fungal cell walls contain (1-3)-beta-D-glucan, a medically significant glucose polymer that has immunosuppressive, mitogenic (i.e. causing mitosis or cell transformation) and inflammatory properties. This mold cell wall component also appears to act synergistically with bacterial endotoxins to produce airway inflammation following inhalation exposure in guinea pigs (Fogelmark et al., 1994).

Under certain metabolic conditions, many fungi produce mycotoxins, natural organic compounds that initiate a toxic response in vertebrates. While some mycotoxins have been found to be associated with hyphae, the primary mode of human exposure to mycotoxins is inhalation of spores and mold-contaminated material. Molds that are important potential producers of toxins indoors are certain species of *Fusarium*, *Penicillium*, and *Aspergillus*. In water-damaged buildings *Stachybotrys chartarum* (a.k.a. *atra*) and *Aspergillus versicolor* may also produce toxic metabolites. A large body of information is available on the human and animal health effects from ingestion of certain mycotoxins (Beasley, 1994; Sorenson, 1989; Smith and Henderson, 1991), but investigators have only recently begun to explore the health implications of inhalation exposure to these substances. Two classes of mycotoxins have been isolated from house dust samples: aflatoxins from some strains of *Aspergillus flavus* and trichothecenes from some species and strains of *Fusarium*, *Cephalosporium*, *Stachybotrys* and *Trichoderma*. In laboratory animals, inhalation of trichothecene mycotoxins causes severe inhibition of protein synthesis and immunosuppression (Beasley, 1994). Several case reports have associated overgrowths of trichothecene-producing fungi with human health effects such as cold and flu-like symptoms, sore throats, headache and general malaise (Croft et al., 1986; Johanning et al., 1993; Nikulin et al., 1994). However, isolation of a toxigenic fungus from a building does not imply the presence of mycotoxin, since the physical conditions necessary for mycotoxin production are very specific, and are often different from those required for growth of the parent mold. Likewise, failure to produce toxins *in vitro* does not mean that a mold known to be toxigenic will not produce toxins in a field situation.

Molds also produce a large number of volatile organic compounds (VOCs). These chemicals are responsible for the musty odors produced by growing molds. There is little evidence that fungal VOCs cause specific human health effects (Batterman, 1995), but the most common VOC, ethanol, is a potent synergizer of many fungal toxins.

Health effects associated with molds

Molds produce acute health effects through toxin-induced [inflammation](#), allergy, or infection. There is no information at this time on the effects of chronic, low dose inhalation exposure to mycotoxins.

Toxin-induced inflammation: Repeated or high exposures to airborne mycotoxins can cause mucous membrane irritation characterized by eye, nose and throat irritation

(Richerson, 1990). When small diameter spores (2-4 μm) are inhaled, they may reach the lung alveoli and induce an inflammatory reaction, creating toxic pneumonitis. Severe toxic pneumonitis can cause fever, flu-like symptoms and fatigue (organic toxic dust syndrome). Hypersensitivity pneumonitis, a particular form of granulomatous lung disease, is a syndrome caused by inhalation of large concentrations of dust containing organic material including fungal spores. It is generally an occupational hazard in agriculture, but has been reported in individuals exposed in the home (Flannigan, *et al.*, 1991). Other symptoms attributed to mycotoxin or fungal-origin VOCs include headache, dizziness, dermatitis, diarrhea and impaired or altered immune function.

Allergy: Indoor fungal allergens probably affect fewer people than do allergens from cats, mites or cockroaches. A significant proportion (10-32%) of asthmatics has been found to be sensitive to fungi. More thorough discussion of fungal allergens is available elsewhere (Horner, *et al.* 1995; Einarsson, *et al.* 1992; Burge, 1985).

Infection: Opportunistic fungal pathogens such as *Aspergillus* are common in indoor air. A normal, healthy individual can probably resist infection by these organisms regardless of dose, although high exposures may cause hypersensitivity pneumonitis. However, any mold that can grow at body temperature can become a pathogen in an immunocompromised host. Individuals undergoing chemotherapy, organ or bone marrow transplantation or those with HIV/AIDS are especially susceptible to invasive infection by *Aspergillus* species.

Some examples of indoor molds, their products and possible health effects are given in Table 2.

Prevention and Control

Although we don't fully understand how or when indoor fungi affect human health, we do have enough evidence to recommend controlling these organisms in indoor environments. Since fungal spores and conidia are ubiquitous, the most effective method of source control is elimination of moisture that supports mold growth. This may involve fixing leaking pipes, windows or roofs, directing rainfall or irrigation drainage away from exterior walls, or increasing insulation. Using fans or opening windows may also be helpful. Ventilation systems, especially those in large commercial buildings, should be properly maintained and examined periodically for microbial contamination.

When underlying moisture sources cannot be readily eliminated, air conditioners and dehumidifiers can help control relative humidity. When using dehumidifiers, water collection traps should be cleaned routinely as these are another source of microbial growth. Visible mold can be removed by disinfection with a chlorine bleach solution. The area being cleaned should be well ventilated, as chlorine itself is volatile and irritating.

Directions of Future Research

There are many gaps in our knowledge of human health effects associated with inhalation exposure to indoor molds. Important research topics include:

- defining how fungal toxins impair immune systems,
- quantifying relationship of dose and duration of exposure to airborne mycotoxins,
- developing efficient methods to identify and analyze mycotoxins in the field,
 - determining effects of varying environmental conditions (substrate temperature, relative humidity, material moisture content) on mycotoxin production, and
 - examining potential human health effects from exposure to combinations of indoor contaminants such as environmental tobacco smoke, VOCs, carbon monoxide, mycotoxins and other microbial components.

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TABLE 1

Selected Important Molds Found in Damp Buildings

Fungal Species	Substrate	Possible Metabolites	Potential Health Effects*
<i>Alternaria alternata</i>	moist window-sills, walls	allergens	asthma, allergy
<i>Aspergillus versicolor</i>	damp wood, wallpaper glue	mycotoxins, VOCs	unknown
<i>Aspergillus fumigatus</i>	house dust, potting soil	allergens	asthma, rhinitis, hypersensitivity pneumonitis
		many mycotoxins	toxic pneumonitis infection**
<i>Cladosporium herbarum</i>	moist window-sills, wood	allergens	asthma, allergy
<i>Penicillium chrysogenum</i>	damp wallpaper, behind paint	mycotoxins	unknown
		VOCs	unknown
<i>Penicillium expansum</i>	damp wallpaper	mycotoxins	nephrotoxicity?
<i>Stachybotrys</i>	heavily wetted	mycotoxins	dermatitis, mucosal

<i>chartarum</i> (<i>atra</i>)	carpet, gypsum board		irritation, immunosuppression
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* specifically from inhalation exposure, based on laboratory animal data

** in immuno-compromised individuals

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