Is Indoor Mold Contamination a Threat to Health?

Dr. Harriet Ammann

The Fungus Among Us

Molds, a subset of the fungi, are ubiquitous on our planet. Fungi are found in every ecological niche, and are necessary for the recycling of organic building blocks that allow plants and animals to live. Included in the group "fungi" are yeasts, molds and mildews, as well as large mushrooms, puffballs and bracket fungi that grow on dead trees. Fungi need external organic food sources and water to be able to grow.

Molds

Molds can grow on cloth, carpets, leather, wood, sheet rock, insulation (and on human foods) when moist conditions exist (Gravesen *et al.*, 1999). Because molds grow in moist or wet indoor environments, it is possible for people to become exposed to molds and their products, either by direct contact on surfaces, or through the air, if mold spores, fragments, or mold products are aerosolized.

Many molds reproduce by making spores, which, if they land on a moist food source, can germinate and begin producing a branching network of cells called hyphae. Molds have varying requirements for moisture, food, temperature and other environmental conditions for growth. Indoor spaces that are wet, and have organic materials that mold can use as a food source, can and do support mold growth. Mold spores or fragments that become airborne can expose people indoors through inhalation or skin contact.

Molds can have an impact on human health, depending on the nature of the species involved, the metabolic products being produced by these species, the amount and duration of individual's exposure to mold parts or products, and the specific susceptibility of those exposed.

Health effects generally fall into four categories. These four categories are allergy, infection, irritation (mucous membrane and sensory), and toxicity.

Allergy

The most common response to mold exposure may be allergy. People who are atopic, that is, who are genetically capable of producing an allergic response, may develop symptoms of allergy when their respiratory system or skin is exposed to mold or mold products to which they have become sensitized. Sensitization can occur in atopic individuals with sufficient exposure.

Allergic reactions can range from mild, transitory responses, to severe, chronic illnesses. The Institute of Medicine (1993) estimates that one in five Americans suffers from allergic rhinitis, the single most common chronic disease experienced by humans. Additionally, about 14 % of the population suffers from allergy-related sinusitis, while 10 to 12% of Americans have

allergically-related asthma. About 9% experience allergic dermatitis. A very much smaller number, less than one percent, suffer serious chronic allergic diseases such as allergic bronchopulmonary aspergillosis (ABPA) and hypersensitivity pneumonitis (Institute of Medicine, 1993). Allergic fungal sinusitis is a not uncommon illness among atopic individuals residing or working in moldy environments. There is some question whether this illness is solely allergic or has an infectious component. Molds are just one of several sources of indoor allergens, including house dust mites, cockroaches, effluvia from domestic pets (birds, rodents, dogs, cats) and microorganisms (including molds).

While there are thousands of different molds that can contaminate indoor air, purified allergens have been recovered from only a few of them. This means that atopic individuals may be exposed to molds found indoors and develop sensitization, yet not be identified as having mold allergy. Allergy tests performed by physicians involve challenge of an individual's immune system by specific mold allergens. Since the reaction is highly specific, it is possible that even closely related mold species may cause allergy, yet that allergy may not be detected through challenge with the few purified mold allergens available for allergy tests. Thus a positive mold allergy test indicates sensitization to an antigen contained in the test allergen (and perhaps to other fungal allergens) while a negative test does not rule out mold allergy for atopic individuals.

Infection

Infection from molds that grow in indoor environments is not a common occurrence, except in certain susceptible populations, such as those with immune compromise from disease or drug treatment. A number of *Aspergillus* species that can grow indoors are known to be pathogens. *Aspergillus fumigatus* (*A. fumigatus*) is a weak pathogen that is thought to cause infections (called aspergilloses) only in susceptible individuals. It is known to be a source of nosocomial infections, especially among immune-compromised patients. Such infections can affect the skin, the eyes, the lung, or other organs and systems. *A. fumigatus* is also fairly commonly implicated in ABPA and allergic fungal sinusitis. *Aspergillus flavus* has also been found as a source of nosocomial infections (Gravesen *et al.*, 1994).

There are other fungi that cause systemic infections, such as *Coccidioides, Histoplasma*, and *Blastomyces.* These fungi grow in soil or may be carried by bats and birds, but do not generally grow in indoor environments. Their occurrence is linked to exposure to wind-borne or animal-borne contamination.

Mucous Membrane and Trigeminal Nerve Irritation

A third group of possible health effects from fungal exposure derives from the volatile compounds (VOC) produced through fungal primary or secondary metabolism, and released into indoor air. Some of these volatile compounds are produced continually as the fungus consumes its energy source during primary metabolic processes. (Primary metabolic processes are those necessary to sustain an individual organism's life, including energy extraction from foods, and the syntheses of structural and functional molecules such as proteins, nucleic acids and lipids). Depending on available oxygen, fungi may engage in aerobic or anaerobic metabolism. They

may produce alcohols or aldehydes and acidic molecules. Such compounds in low but sufficient aggregate concentration can irritate the mucous membranes of the eyes and respiratory system.

Just as occurs with human food consumption, the nature of the food source on which a fungus grows may result in particularly pungent or unpleasant primary metabolic products. Certain fungi can release highly toxic gases from the substrate on which they grow. For instance, one fungus growing on wallpaper released the highly toxic gas arsine from arsenic containing pigments (Gravesen, *et al.*, 1994).

Fungi can also produce secondary metabolites as needed. These are not produced at all times since they require extra energy from the organism. Such secondary metabolites are the compounds that are frequently identified with typically "moldy" or "musty" smells associated with the presence of growing mold. However, compounds such as pinene and limonene that are used as solvents and cleaning agents can also have a fungal source. Depending on concentration, these compounds are considered to have a pleasant or "clean" odor by some people. Fungal volatile secondary metabolites also impart flavors and odors to food. Some of these, as in certain cheeses, are deemed desirable, while others may be associated with food spoilage. There is little information about the advantage that the production of volatile secondary metabolites imparts to the fungal organism. The production of some compounds is closely related to sporulation of the organism. "Off" tastes may be of selective advantage to the survival of the fungus, if not to the consumer.

In addition to mucous membrane irritation, fungal volatile compounds may impact the "common chemical sense" which senses pungency and responds to it. This sense is primarily associated with the trigeminal nerve (and to a lesser extent the vagus nerve). This mixed (sensory and motor) nerve responds to pungency, not odor, by initiating avoidance reactions, including breath holding, discomfort, or paresthesias, or odd sensations, such as itching, burning, and skin crawling. Changes in sensation, swelling of mucous membranes, constriction of respiratory smooth muscle, or dilation of surface blood vessels may be part of fight or flight reactions in response to trigeminal nerve stimulation. Decreased attention, disorientation, diminished reflex time, dizziness and other effects can also result from such exposures (Otto *et al.*, 1989)

It is difficult to determine whether the level of volatile compounds produced by fungi influence the total concentration of common VOCs found indoors to any great extent. A mold-contaminated building may have a significant contribution derived from its fungal contaminants that is added to those VOCs emitted by building materials, paints, plastics and cleaners. Miller and co-workers (1988) measured a total VOC concentration approaching the levels at which Otto *et al.*, (1989) found trigeminal nerve effects.

At higher exposure levels, VOCs from any source are mucous membrane irritants, and can have an effect on the central nervous system, producing such symptoms as headache, attention deficit, inability to concentrate or dizziness.

Adverse Reactions to Odor

Odors produced by molds may also adversely affect some individuals. Ability to perceive odors

and respond to them is highly variable among people. Some individuals can detect extremely low concentrations of volatile compounds, while others require high levels for perception. An analogy to music may give perspective to odor response. What is beautiful music to one individual is unbearable noise to another. Some people derive enjoyment from odors of all kinds. Others may respond with headache, nasal stuffiness, nausea or even vomiting to certain odors including various perfumes, cigarette smoke, diesel exhaust or moldy odors. It is not know whether such responses are learned, or are time-dependent sensitization of portions of the brain, perhaps mediated through the olfactory sense (Bell, *et al.*, 1993a; Bell *et al.*, 1993b), or whether they serve a protective function. Asthmatics may respond to odors with symptoms.

Toxicity

Molds can produce other secondary metabolites such as antibiotics and mycotoxins. Antibiotics are isolated from mold (and some bacterial) cultures and some of their bacteriotoxic or bacteriostatic properties are exploited medicinally to combat infections.

Mycotoxins are also products of secondary metabolism of molds. They are not essential to maintaining the life of the mold cell in a primary way (at least in a friendly world), such as obtaining energy or synthesizing structural components, informational molecules or enzymes. They are products whose function seems to be to give molds a competitive advantage over other mold species and bacteria. Mycotoxins are nearly all cytotoxic, disrupting various cellular structures such as membranes, and interfering with vital cellular processes such as protein, RNA and DNA synthesis. Of course they are also toxic to the cells of higher plants and animals, including humans.

Mycotoxins vary in specificity and potency for their target cells, cell structures or cell processes by species and strain of the mold that produces them. Higher organisms are not specifically targeted by mycotoxins, but seem to be caught in the crossfire of the biochemical warfare among mold species and molds and bacteria vying for the same ecological niche.

Not all molds produce mycotoxins, but numerous species do (including some found indoors in contaminated buildings). Toxigenic molds vary in their mycotoxin production depending on the substrate on which they grow (Jarvis, 1990). The spores, with which the toxins are primarily associated, are cast off in blooms that vary with the mold's diurnal, seasonal and life cycle stage (Burge, 1990; Yang, 1995). The presence of competitive organisms may play a role, as some molds grown in monoculture in the laboratory lose their toxic potency (Jarvis, 1995). Until relatively recently, mold poisons were regarded with concern primarily as contaminants in foods.

More recently concern has arisen over exposure to multiple mycotoxins from a mixture of mold spores growing in wet indoor environments. Health effects from exposures to such mixtures can differ from those related to single mycotoxins in controlled laboratory exposures. Indoor exposures to toxigenic molds resemble field exposures of animals more closely than they do controlled experimental laboratory exposures. Animals in controlled laboratory exposures are healthy, of the same age, raised under optimum conditions, and have only the challenge of known doses of a single toxic agent via a single exposure route. In contrast, animals in field exposures are of mixed ages, and states of health, may be living in less than optimum

environmental and nutritional conditions, and are exposed to a mixture of toxic agents by multiple exposure routes. Exposures to individual toxins may be much lower than those required to elicit an adverse reaction in a small controlled exposure group of ten animals per dose group. The effects from exposure may therefore not fit neatly into the description given for any single toxin, or the effects from a particular species, of mold.

Field exposures of animals to molds (in contrast to controlled laboratory exposures) show effects on the immune system as the lowest observed adverse effect. Such immune effects are manifested in animals as increased susceptibility to infectious diseases (Jakab *et al.*, 1994). It is important to note that almost all mycotoxins have an immunosuppressive effect, although the exact target within the immune system may differ. Many are also cytotoxic, so that they have route of entry effects that may be damaging to the gut, the skin or the lung. Such cytotoxicity may affect the physical defense mechanisms of the respiratory tract, decreasing the ability of the airways to clear particulate contaminants (including bacteria or viruses), or damage alveolar macrophages, thus preventing clearance of contaminants from the deeper lung. The combined result of these activities is to increase the susceptibility of the exposed person to infectious disease, and to reduce his defense against other contaminants. They may also increase susceptibility to cancer

Because indoor samples are usually comprised of a mixture of molds and their spores, it has been suggested that a general test for cytotoxicity be applied to a total indoor sample to assess the potential for hazard as a rough assessment (Gareis, 1995).

The following summary of toxins and their targets is adapted from Smith and Moss (1985), with a few additions from the more recent literature. While this compilation of effects does not describe the effects from multiple exposures, which could include synergistic effects, it does give a better idea of possible results of mycotoxin exposure to multiple molds indoors.

- Vascular system (increased vascular fragility, hemorrhage into body tissues, or from lung, e.g., aflatoxin, satratoxin, roridins).

- Digestive system (diarrhea, vomiting, intestinal hemorrhage, liver effects, i.e., necrosis, fibrosis: aflatoxin; caustic effects on mucous membranes: T-2 toxin; anorexia: vomitoxin.

- Respiratory system: respiratory distress, bleeding from lungs e.g., trichothecenes.

- Nervous system, tremors, incoordination, depression, headache, e.g., tremorgens, trichothecenes.

- Cutaneous system : rash, burning sensation sloughing of skin, photosensitization, e.g., trichothecenes.

- Urinary system, nephrotoxicity, e.g. ochratoxin, citrinin.

- Reproductive system; infertility, changes in reproductive cycles, e.g. T-2 toxin, zearalenone.

- Immune system: changes or suppression: many mycotoxins.

It should be noted that not all mold genera have been tested for toxins, nor have all species within a genus necessarily been tested. Conditions for toxin production varies with cell and diurnal and seasonal cycles and substrate on which the mold grows, and those conditions created for laboratory culture may differ from those the mold encounters in its environment.

Toxicity can arise from exposure to mycotoxins via inhalation of mycotoxin-containing mold spores or through skin contact with the toxigenic molds (Forgacs, 1972; Croft *et al.*, 1986; Kemppainen *et al.*, 1988 -1989). A number of toxigenic molds have been found during indoor air quality investigations in different parts of the world. Among the genera most frequently found in numbers exceeding levels that they reach outdoors are *Aspergillus*, *Penicillium*, *Stachybotrys*, and *Cladosporium* (Burge, 1986; Smith *et al.*, 1992; Hirsh and Sosman, 1976; Verhoeff *et al.*, 1992; Miller *et al.*, 1988; Gravesen *et al*., 1999). *Penicillium, Aspergillus* and *Stachybotrys* toxicity, especially as it relates to indoor exposures, will be discussed briefly in the paragraphs that follow.

Penicillium

Penicillium species have been shown to be fairly common indoors, even in clean environments, but certainly begin to show up in problem buildings in numbers greater than outdoors (Burge, 1986; Miller *et al*., 1988; Flannigan and Miller, 1994). Spores have the highest concentrations of mycotoxins, although the vegetative portion of the mold, the mycelium, can also contain the poison. Viability of spores is not essential to toxicity, so that the spore as a dead particle can still be a source of toxin.

Important toxins produced by penicillia include nephrotoxic citrinin, produced by *P. citrinum, P. expansum* and *P. viridicatum;* nephrotoxic ochratoxin, from *P. cyclopium* and *P. viridicatum*, and patulin, cytotoxic and carcinogenic in rats, from *P. expansum* (Smith and Moss, 1985).

Aspergillus

Aspergillus species are also fairly prevalent in problem buildings. This genus contains several toxigenic species, among which the most important are, *A. parasiticus, A. flavus*, and *A. fumigatus*. Aflatoxins produced by the first two species are among the most extensively studied mycotoxins. They are among the most toxic substances known, being acutely toxic to the liver, brain, kidneys and heart, and with chronic exposure, potent carcinogens of the liver. They are also teratogenic (Smith and Moss, 1985; Burge, 1986). Symptoms of acute aflatoxicosis are fever, vomiting, coma and convulsions (Smith and Moss, 1985). *A. flavus* is found indoors in tropical and subtropical regions, and occasionally in specific environments such as flowerpots. *A. fumigatus* has been found in many indoor samples. A more common aspergillus species found in wet buildings is *A. versicolor*, where it has been found growing on wallpaper, wooden floors, fibreboard and other building material. *A. versicolor* does not produce aflatoxins, but does produce a less potent toxin, sterigmatocystin, an aflatoxin precursor (Gravesen *et al*., 1994). While symptoms of aflatoxin exposure through ingestion are well described, symptoms of exposure such as might occur in most moderately contaminated buildings are not know, but are

undoubtedly less severe due to reduced exposure. However, the potent toxicity of these agents advise that prudent prevention of exposures are warranted when levels of aspergilli indoors exceed outdoor levels by any significant amount. *A. fumigatus* has been found in many indoor samples. This mold is more often associated with the infectious disease aspergillosis, but this species does produce poisons for which only crude toxicity tests have been done (Betina, 1989). Recent work has found a number of tremorgenic toxins in the conidia of this species (Land *et al*., 1994). *A. ochraceus* produces ochratoxins (also produced by some penicillia as mentioned above). Ochratoxins damage the kidney and are carcinogenic (Smith and Moss, 1985).

Stachybotrys chartarum (atra)

Stachybotrys chartarum (atra) has been much discussed in the popular press and has been the subject of a number of building related illness investigations. It is a mold that is not readily measured from air samples because its spores, when wet, are sticky and not easily aerosolized. Because it does not compete well with other molds or bacteria, it is easily overgrown in a sample, especially since it does not grow well on standard media (Jarvis, 1990). Its inability to compete may also result in its being killed off by other organisms in the sample mixture. Thus, even if it is physically captured, it will not be viable and will not be identified in culture, even though it is present in the environment and those who breathe it can have toxic exposures. This organism has a high moisture requirement, so it grows vigorously where moisture has accumulated from roof or wall leaks, or chronically wet areas from plumbing leaks. It is often hidden within the building envelope. When *S. chartarum* is found in an air sample, it should be searched out in walls or other hidden spaces, where it is likely to be growing in abundance. This mold has a very low nitrogen requirement, and can grow on wet hay and straw, paper, wallpaper, ceiling tiles, carpets, insulation material (especially cellulose-based insulation). It also grows well when wet filter paper is used as a capturing medium.

S. chartarum has a well-known history in Russia and the Ukraine, where it has killed thousands of horses, which seem to be especially susceptible to its toxins. These toxins are macrocyclic trichothecenes. They cause lesions of the skin and gastrointestinal tract, and interfere with blood cell formation. (Sorenson, 1993). Persons handling material heavily contaminated with this mold describe symptoms of cough, rhinitis, burning sensations of the mouth and nasal passages and cutaneous irritation at the point of contact, especially in areas of heavy perspiration, such as the armpits or the scrotum (Andrassy *et al*., 1979).

One case study of toxicosis associated with macrocyclic trichothecenes produced by *S. chartarum* in an indoor exposure, has been published (Croft *et al.*, 1986), and has proven seminal in further investigations for toxic effects from molds found indoors. In this exposure of a family in a home with water damage from a leaky roof, complaints included (variably among family members and a maid) headaches, sore throats, hair loss, flu symptoms, diarrhea, fatigue, dermatitis, general malaise, psychological depression. (Croft *et al*, 1986; Jarvis, 1995).

Johanning, (1996) in an epidemiological and immunological investigation, reports on the health status of office workers after exposure to aerosols containing *S. chartarum.* Intensity and duration of exposure was related to illness. Statistically significant differences for more exposed groups were increased lower respiratory symptoms, dermatological, eye and constitutional

symptoms, chronic fatigue, and allergy history. Duration of employment was associated with upper respiratory, skin and central nervous system disorders. A trend for frequent upper respiratory infections, fungal or yeast infections, and urinary tract infections was also observed. Abnormal findings for components of the immune system were quantified, and it was concluded that higher and longer indoor exposure to *S. chartarum* results in immune modulation and even slight immune suppression, a finding that supports the observation of more frequent infections.

Three articles describing different aspects of an investigation of acute pulmonary hemorrhage in infants, including death of one infant, have been published recently, as well as a CDC evaluation of the investigation (Monta» a *et al*., 1997; Etzel *et al*., 1998; Jarvis *et al.*, 1998; MMWR, 2000; CDC, 1999). The infants in the Cleveland outbreak were reported with pulmonary hemosiderosis, a sign of an uncommon of lung disease that involves pulmonary hemorrhage. *Stachybotrys chartarum* was shown to have an association with acute pulmonary bleeding. Additional studies are needed to confirm association and establish causality.

Animal experiments in which rats and mice were exposed intranasally and intratracheally to toxic strains of *S. chartarum*, demonstrated acute pulmonary hemorrhage (Nikkulin *et al*. 1996). A number of case studies have been more recently published. One involving an infant with pulmonary hemorrhage in Kansas, reported significantly elevated spore counts of *Aspergillus/Penicillium* in the patient's bedroom and in the attic of the home. *Stachybotrys* spores were also found in the air of the bedroom, and the source of the spores tested highly toxigenic (Flappan *et al*., 1999). In another case study in Houston, *Stachybotrys* was isolated from bronchopulmonary lavage fluid of a child with pulmonary hemorrhage. (Elidemir *et al*., 1999), as well as recovered from his water damaged-home. The patient recovered upon removal and stayed well after return to a cleaned home. Another case study reported pulmonary hemorrhage in an infant during induction of general anesthesia. The infant was found to have been exposed to *S. chartarum* prior to the anesthetic procedure (Tripi *et al*., 2000). Still another case describes pulmonary hemorrhage in an infant whose home contained toxigenic species of *Penicillium* and *Trichoderma* (a mold producing trichothecene poisons similar to the ones produced by *S. chartarum*) as well as tobacco smoke (Novotny and Dixit, 2000)

Toxicologically, *S. chartarum* can produce extremely potent trichothecene poisons, as evidenced by one-time lethal doses in mice (LD_{50}) as low as 1.0 to 7.0 mg/kg, depending on the toxin and the exposure route. Depression of immune response, and hemorrhage in target organs are characteristic for animals exposed experimentally and in field exposures (Ueno, 1980; Jakab *et al*., 1994).

While there are insufficient studies to establish cause and effect relationships between *Stachybotrys* exposure indoors and illness, including acute pulmonary bleeding in infants, toxic endpoints and potency for this mold are well described. What is less clear, and has been difficult to establish, is whether exposures indoors are of sufficient magnitude to elicit illness resulting from toxic exposure.

Some of these difficulties derive from the nature of the organisms and the toxic products they produce and varying susceptibilities among those exposed. Others relate to problems common to

retrospective case control studies. Some of the difficulties in making the connection between toxic mold exposures and illness are discussed below.

Limitations in Sampling Methodology, Toxicology, and Epidemiology of Toxic Mold Exposure

Some of the difficulties and limitations encountered in establishing links between toxic mold contaminated buildings and illness are listed here:

- Few toxicological experiments involving mycotoxins have been performed using inhalation, the most probable route for indoor exposures. Defenses of the respiratory system differ from those for ingestion (the route for most mycotoxin experiments). Experimental evidence suggests the respiratory route to produce more severe responses than the digestive route (Cresia *et al.*, 1987)

- Effects from low level or chronic low level exposures, or ingestion exposures to mixtures of mycotoxins, have generally not been studied, and are unknown. Effects from high level, acute sub-acute and sub-chronic ingestion exposures to single mycotoxins have been studied for many of the mycotoxins isolated. Other mycotoxins have only information on cytotoxicity or *in vitro* effects.

- Effects of multiple exposures to mixtures of mycotoxins in air, plus other toxic air pollutants present in all air breathed indoors, are not known.

- Effects of other biologically active molecules, having allergic or irritant effects, concomitantly acting with mycotoxins, are not known.

- Measurement of mold spores and fragments varies, depending on instrumentation and methodology used. Comparison of results from different investigators is rarely, if ever, possible with current state of the art.

- While many mycotoxins can be measured in environmental samples, it is not yet possible to measure mycotoxins in human or animal tissues. For this reason exposure measurements rely on circumstantial evidence such as presence of contamination in the patient's environment, detection of spores in air, combined with symptomology in keeping with known experimental lesions caused by mycotoxins, to establish an association with illness.

- Response of individuals exposed indoors to complex aerosols varies depending on their age, gender, state of health, and genetic make-up, as well as degree of exposure.

- Microbial contamination in buildings can vary greatly, depending on location of growing organisms, and exposure pathways. Presence in a building alone does not constitute exposure.

- Investigations of patients' environments generally occur after patients have become ill, and do not necessarily reflect the exposure conditions that occurred during development of the

illness. All cases of inhalation exposure to toxic agents suffer from this deficit. However exposures to chemicals not generated biologically can sometimes be re-created, unlike those with active microbial growth. Indoor environments are dynamic ecosystems that change over time as moisture, temperature, food sources and the presence of other growing microorganisms change. Toxin production particularly changes with age of cultures, stage of sporulation, availability of nutrients, moisture, and the presence of competing organisms. After-the-fact measurements of environmental conditions will always reflect only an estimate of exposure conditions at the time of onset of illness. However, presence of toxigenic organisms, and their toxic products, are indicators of putative exposure, which together with knowledge of lesions and effects produced by toxins found, can establish association.

Conclusions and Recommendations

Prudent public health practice then indicates removal from exposure through clean up or remediation, and public education about the potential for harm. Not all species within these genera are toxigenic, but it is prudent to assume that when these molds are found in excess indoors that they are treated as though they are toxin producing. It is not always cost effective to measure toxicity, so cautious practice regards the potential for toxicity as serious, aside from other health effects associated with excessive exposure to molds and their products. It is unwise to wait to take action until toxicity is determined after laboratory culture, especially since molds that are toxic in their normal environment may lose their toxicity in laboratory monoculture over time (Jarvis, 1995) and therefore may not be identified as toxic. While testing for toxins is useful for establishing etiology of disease, and adds to knowledge about mold toxicity in the indoor environment, prudent public health practice might advise speedy clean-up, or removal of a heavily exposed populations from exposure as a first resort.

Health effects from exposures to molds in indoor environments can result from allergy, infection, mucous membrane and sensory irritation and toxicity alone, or in combination. Mold growth in buildings (in contrast to mold contamination from the outside) always occurs because of unaddressed moisture problems. When excess mold growth occurs, exposure of individuals, and remediation of the moisture problem must be addressed.

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Please note: This document was last reviewed in 2003. For more recent information on mold and health affects please read:

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Edited by Bradley Prezant, Donald M. Weekes, and J. David Miller
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Harriet M. Ammann, Ph.D., DABT
Senior Toxicologist and Principal
Ammann Toxicology Consulting LLC
Olympia Washington
Affiliate Associate Professor, Department of Environmental Health
University of Washington School of Public Health and Community Medicine

Harriet M. Ammann, a diplomate of the American Board of Toxicology since 1989, served as senior toxicologist for Department of Health's Office of Environmental Health Assessment for twelve years, and as senior toxicologist for the Washington Department of Ecology Air Program for four years, before retiring from state service in September of 2006. She has participated in evaluations of schools and public buildings with air quality problems, and has presented on toxic effects from air contaminants, indoors and out, effect on sensitive populations, and other health issues throughout the state.

Dr. Ammann served as vice-chair of the Bioaerosols Committee of the American Conference of Governmental Industrial Hygienists (ACGIH) from 1997-2003. She served on the Institute of Medicine (National Academies of Science) Committee on Damp Indoor Spaces and Health and was co-author of the Committee's report ***Damp Indoor Spaces and Health***, focusing her efforts primarily on the toxicity chapter, and damp buildings public health chapters (2004). She authored the chapter on Microbial VOCs in the ACGIH book, ***Bioaerosols, Assessment and Control,*** and contributed to chapters on Data Evaluation and Fungal Toxins and β-D-Glucans. She also served as assistant editor to the book. Additionally she has published and presented on mold toxicity to national and international conferences. Most recently she was invited to present on mycotoxin toxicity and exposure at the 26th International Mycotoxin Research Conference in Dortmund, Germany (2005), and to the Tenth International Congress of Toxicology Satellite Conference on Mold Toxicity in Kuopio Finland (July 2004) and to the NIEHS National Meeting on Mold-Related Health Effects in Washington DC (2004), and for the 5th International Conference on Bioaerosols, Fungi, Bacteria, Mycotoxins and Human Health (September 2003). She was also invited to deliver the keynote address *Air Pollution and Asthma* for the environmental health section of the CDC National Asthma Conference in Atlanta (April 2004).

She was a contributing author to Chapter 1, and co-editor of Section 1 of the 2008 AIHA book: **Recognition, Evaluation, and Control of Indoor Mold.**