

## HEALTH ISSUES WITH MOULDS

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### Summary

The health effects of moulds growing within the built environment are viewed differently depending on one's point of view. Home owners, contractors, lawyers, and physicians all receive and process information from different sources. This paper explores some of the issues surrounding the health effects of moulds.

### Introduction

Humans have co-evolved with moulds. Changes in building construction practice, and natural disasters such as flooding have focused attention on the relationship of moulds and human habitats. Moulds have literally become a growing business. Hard on the heels of insurance companies eliminating home coverage for water damage comes toxic mould legislation in several U.S. states. Moulds can affect human health, but in addition to biomedical models of host-disease relationships, public perception of the relationship and the cost of moulds in homes, offices, and schools may also affect health. Sources of information regarding the magnitude of health outcomes may differ for different segments of society.

### Home and building owners

It is impossible to separate the effects of stress due to financial burden from any discussion of the "true" health effects of airborne moulds. In the United States very large financial settlements have served to corroborate the perception of the dangers of moulds in homes. Homeowners may access information from the internet, with the added bonus that legal advice is close at hand. All one needs to do is access <http://www.atoxicmoldattorneyforyou.com> to find a firm close to home.

Within the last two years several states have passed toxic mould legislation (Louisiana, Montana, Oklahoma, Oregon, South Carolina, Texas, and Virginia)<sup>1</sup>. A common feature of these laws is the necessity of disclosure of mould or water damage during real estate transactions. Not all states have addressed parallel legislation requiring certification for mould inspectors and remediators. Some states have enabled task forces to research the relationship of moulds and health (Illinois, Massachusetts, Oklahoma, Pennsylvania and Rhode Island).<sup>1</sup> In the absence of home insurance for water and mould damage, the *Melinda Bill* has been introduced in the House of Representatives that would provide national insurance and medicaid coverage for victims of mould.<sup>2</sup> No objective criteria defining a hazardous level of mould have yet been universally accepted. Thus, owners of buildings must rely on the expert judgment of the mould inspector.

## Consultants and Contractors

Most contractors rely on the guidance of the New York Protocol<sup>3</sup> or a variation of the protocol to determine the level of protection required for mould remediation projects. In recognition that the greater the amount of visible mould

present on building materials must result in a greater amount of fungal products released during renovations, most protocols define levels of protection for workers and occupants based on area of mould damage. The lowest level is defined as 10 ft<sup>2</sup>. Larger projects require full isolation under negative pressure and monitoring throughout the project, very similar to asbestos abatement requirements. Unlike asbestos abatement, clearance levels are not well defined, and usually depend on expert judgment of the contractor.

Guidance in the absence of visible mould differs between jurisdictions and often depends on the results of air sampling. Action levels vary from 50 CFU/m<sup>3</sup> to 1000 CFU/m<sup>3</sup>.<sup>4</sup> There are no standards for sampling equipment or protocols.

## Medical evidence of health effects

Much of the controversy regarding health effects of moulds focuses on the range of purported symptoms caused by exposure to mould. Medical models require a clear route of exposure to sufficient quantities of the offending agent to initiate the disease process. Several diseases are recognized as fulfilling these criteria. Allergic alveolitis (hypersensitivity pneumonitis) was recognized in 1713 by Ramazzini, a physician who documented many occupational diseases. Allergic alveolitis causes lung scarring through host-antigen interaction by fungal spores such as *Aspergillus*, *Penicillium* or the bacterial spores of *Thermoactinomyces*. This condition is an outcome of repeated exposure to antigen and the action of host macrophage activation. Although allergic alveolitis is often thought of as only an occupational hazard, there are several case studies in the literature of domestic exposures which include *Serpula lacrymans*, *Paecilomyces*, *Aspergillus fumigatus*, *Epicoccum nigrum*, *Thermo-actinomyces* and *Trichosporon*. Another disease, but which is normally self limiting, is Organic Dust Toxic Syndrome (ODTS). ODTS results from high exposure to fungal spores, bacteria, and endotoxin.<sup>5-8</sup>

Some fungal species are pathogenic because they are able to grow at body temperature. Pathogenic fungi which have been found associated with water damaged environments include *Aspergillus fumigatus*. *A. fumigatus* is a leading cause of death in persons who have damaged immune systems (e.g. bone marrow transplants, AIDS, organ transplantations). *A. fumigatus* is among several fungi implicated in cases of chronic sinusitis. Other fungi may not be pathogenic *per se*, but may stimulate formation of reactive immunoglobulins which result in allergic reactions in a portion of the population. Asthma is one example of disease exacerbated by fungal exposures due to the action of reactive immunoglobulins in the bronchial passages. In addition to specific immune reactions to antigens, fungal spores contain polysaccharide structural compounds such as 1-3 or 1-6 beta D glucan, which have been shown to have immunoregulating effects which are independent of immunoglobulins. Recent studies have shown that the age at

which the host is first exposed to fungal antigens may influence the development of the immune system. For example, the immune system of babies may be more prone to develop hyper-reactive “T” cells if, at a critical time window of the maturation process, the “T” cells are exposed to fungal antigens. This hyper-reactivity may then persist for life.

Many fungi are capable of producing mycotoxins as secondary metabolites under competitive or stressful growing conditions. The effects of ingested mycotoxins are well documented (aflatoxins, trichothecenes, ergot alkaloids). The action and toxic dose of inhaled mycotoxins are not as well understood, and remain controversial as a major mechanism of fungal disease in domestic exposures. More research is needed in order to develop measurement methods for airborne mycotoxins in domestic environments. Other metabolic products secondary to fungal growth are volatile compounds including alcohols, aldehydes, esters, hydrocarbons, terpenes, ketones, and organic acids. These compounds are well recognized as the “musty” odour of mouldy materials. These volatile compounds may be responsible for some of the respiratory or mucous membrane irritation experienced by occupants of water damaged buildings.

Sufficient epidemiological evidence exists through multiple studies conducted around the world to say that exposure to fungi in indoor environments contribute to symptoms of cough, wheeze, asthma, increased cases of bronchitis, upper respiratory diseases, and eye irritation.<sup>9</sup> There are gaps in knowledge for which there is insufficient support at present to support a causative relationship between fungal exposures and immunodeficiency disease, neurotoxicity, or pulmonary hemosiderosis.<sup>5</sup>

Table 1. Epidemiologic evidence of adverse health outcomes associated with living in water-damaged buildings<sup>10</sup>.

Health indicator	Dampness/mould		Flood		Moisture	
	Odds Ratio	95% CI	Odds Ratio	95% CI	Odds Ratio	95% CI
<b>Cough</b>	<b>1.9</b>	1.6 – 2.2	<b>1.4</b>	1.2 – 1.7	<b>1.9</b>	1.6 – 2.3
<b>Wheeze</b>	<b>1.6</b>	1.4 – 1.8	<b>1.3</b>	1.1 – 1.4	<b>1.7</b>	1.5 – 2.0
<b>Asthma</b>	<b>1.5</b>	1.2 – 1.7	<b>1.3</b>	1.1 – 1.6	<b>1.6</b>	1.3 – 1.9
<b>Bronchitis</b>	<b>1.3</b>	1.2 – 1.5	<b>1.1</b>	1.0 – 1.3	<b>1.5</b>	1.3 – 1.7
<b>Upper respiratory</b>	<b>1.5</b>	1.4 – 1.6	<b>1.3</b>	1.2 – 1.4	<b>1.6</b>	1.4 – 1.9
<b>Eye irritation</b>	<b>1.5</b>	1.3 – 1.8	<b>1.4</b>	1.2 – 1.7	<b>1.6</b>	1.4 – 1.9
<b>Non-respiratory symptoms</b>	<b>1.4</b>	1.3 – 1.6	<b>1.3</b>	1.1 – 1.4	<b>1.5</b>	1.3 – 1.6

### Building materials and control of fungal growth

The challenge remains to limit exposure to fungal products in the indoor environment. A multidisciplinary approach will be necessary which incorporates building design, engineering, and construction and materials. If the experience of Vancouver, British Columbia, is an example, steel and concrete high rise structures are now showing signs of significant water damage, and will require billions of dollars to repair. Dry building

materials do not permit mould growth. Most building materials, if kept continuously wet, will provide substrate or water for surface growth of fungi and bacteria.

Research through interdisciplinary teams may help restore consumer confidence in wood and other building products. Education of consumers, inspectors and renovators is a priority. Aesthetics and financial burden may be confused with ill health, while at the same time, members of the public with immunological sensitivities may only obtain relief from symptoms by complete avoidance of their places of work or occupancy. Anti-fungal agents used to treat building materials must also be safe for long term exposures to susceptible occupants of buildings which may include the very young and the very old. Interdisciplinary teams are well placed to develop protocols which would better quantify mould growth, which in turn would help epidemiologists understand the effects of dose and routes of exposure in health studies.

### **Conclusion**

While the evidence is inconclusive that mould in homes and offices is truly the modern equivalent of the plagues of Egypt, there is more than enough evidence that living or working in water-damaged or mouldy buildings can have significant health effects. In most cases, removal from the exposure results in a return to health. More troubling is the emerging evidence that early childhood exposure to certain antigens may predispose the developing immune system to life-long hypersensitivity. This evidence taken with the profound effect the news media has had on public perception may result in a misperception by the public that common building materials such as lumber may be a contributing factor to ill health. More education is needed for the public and for professionals such as architects, building engineers, and contractors. The public may benefit from instructions on proactive maintenance of their homes. Architects, building engineers and contractors should be encouraged to improve attention to detailing which may ensure that building materials are allowed to remain dry. Interdisciplinary research is clearly required to understand the complex relationship between all of the constituents present in indoor air and the health of occupants.

### **Literature**

1. MoldUpdate.com. <http://www.moldupdate.com/legislation.htm>. Accessed October 2004.
2. Congressman John Conyers, Jr. Home Page. <http://house.gov/conyers/mold.htm>. Accessed October 2004.
3. New York City Department of Health & Mental Hygiene. 2002. Guidelines on Assessment and Remediation of Fungi in Indoor Environments. <http://www.ci.nyc.ny.us/html/doh/home.html>. Accessed October 2004.
4. Rao, CY, Burge HA, Brain JD. Mycopathologia 149(1): 27-34. 2000.
5. Institute of Medicine. Damp Indoor Spaces and Health. National Academies Press. 2004
6. Gravesen S, Frisvad JC, Samson RA. Microfungi. Munksgaard 1994.
7. Rylander R Lin R-H. Toxicology 152: 47 – 52. 2000.

8. Simpson JC, Niven RM, Pickering CA, Fletcher AM, Oldham LA, Francis HM. *Occup Environ Med* 55: 668-672. 1998.
9. Bornehag C-G, Blomquist G, Gyntelberg F, Järholm B, Malmberg P, Nordvall L, Nielsen A, Pershagen G, Sundell J. *Indoor Air* 11:72 – 86. 2001.
10. Dales, RE, Zwanenburg h, Burnett R, Franklin, CA. *Am J Epidemiology* 134: 196-203. 1991.