

Draft 4

Hazard and Risk overview of mould in Water Damaged Buildings

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Thanks

Jeff

1. Health Effects and presumed Safe Levels of Exposure to Mould

1.1. Water Damaged Buildings WDBs can be expected to be a health risk from a variety of recognised contaminants¹ some of which may be considered to be toxic and almost always allergenic to some degree and known or expected to affect 40% of the population² who are recognised as atopic.

1.2. Typical health effects from WDB exposure

- 1.2.1. Respiratory³
- 1.2.2. Skin⁴
- 1.2.3. Gastrointestinal⁵
- 1.2.4. Fatigue and Neuropsychiatric⁶
- 1.2.5. Cancers⁷
- 1.2.6. Reproductive issue⁸
- 1.2.7. Rheumatic and other immune disease⁹
- 1.2.8. Suppression of immune system¹⁰
- 1.2.9. Cardiovascular¹¹

1.3. The degree of risk will revolve around:

- 1.3.1. High levels of exposure over a short period

¹ Jack D. Thrasher DOI: 10.1177/0748233709348386
tih.sagepub.com

² Health effects associated with mold Council on Scientific Affairs Volume 45 issue 5 page 470

³ Wald et al 1997

⁴ IOM Chapter 5 page 244

⁵ Dales et al 1991

⁶ Rylander 1998b Kostiken et al Pirhonen et al 1996

⁷ Rao 2000 et al. Van Vleet et al 2002

⁸ IOM Chapter 5 page 251

⁹ Franceschini et al 1999 and Myllykangas-Luoujarvi et al 2002

¹⁰ Etzel 2002 Jakab et al 1994 and Pier Mcloughlin 1985

¹¹ ACGIH 24.2.2.4

- 1.3.2. Low levels of exposure over a long period
 - 1.3.3. High levels of exposure over a long period
 - 1.3.4. Resilience or strength of immune system and genetics
- 1.4. The quantity of biological activity and in particular mould growth may be subject to contusive environmental conditions being present. Mould growth in particular will depend on the substrate it colonises and time. Moulds are capable of producing large quantities of spores within a day in moist conditions¹² and massive fungal growth within a week following fire fighting efforts.¹³

2. The contaminants may include:

- 2.1.1. Toxic mould spores
- 2.1.2. Non toxic but allergenic mould spores
- 2.1.3. Endotoxins¹⁴, the allergenic cell wall fragments of gram negative bacteria
- 2.1.4. Hyphae¹⁵ fragments both allergenic and possibly toxic
- 2.1.5. Bacteria¹⁶ and their toxins
- 2.1.6. β Glucans 1-3¹⁷ (allergens)
- 2.1.7. MVOCs¹⁸
- 2.1.8. Mycotoxins from household dust not containing spores¹⁹
- 2.1.9. VOCs such as formaldehyde²⁰ (released from damp building materials)

3. In the absence of any research into the health effects or data on exposure to the compound contaminants which may be present; we can only make broad and unsubstantiated assessments of the possible synergistic effects. While this cannot be described as scientific it can be recognised as normal risk and hazard assessment and case studies²¹ and review of scientific evidence identifies these issues²². Normally accepted risk protocol will require the assessment of all possible, likely or presumed hazards and where absence of evidence exists, it would be normal practice to assume the highest threat or hazard level exists.

<http://www.buildingforensics.co.uk/portals/15/pdf/14PCAand%20Mould%20review%20D2.pdf>

3.1. Hazard and risk

The criteria of risk and hazard assessments are identified as:

- 3.1.1. **Hazard** *anything that can cause harm*
- 3.1.2. **Risk**, *the chance or likelihood of harm*

4. Combined and Synergistic Health Hazards

¹² Pasanen et al 1992

¹³ Rautiala et al 2002

¹⁴ Doves and Heedrik 1997 Milton 1999 Ryland literature review 2002

¹⁵ Gorny et al 2002 Englehart et al 2002

¹⁶ Anderson et al 1998 Jussila et al 2001

¹⁷ Rylands literature review 2002 Fogelmark et al 1994

¹⁸ Korbi2001 and Smedje et al 1996

¹⁹ Englehart et al 2002 Gorney et al 2002 Larsen and Frisvad 1994

²⁰ Van Netten et al 1989 anderson et al 1975 Sjoberg and Nilsson 2002

²¹ Journal of Environmental and Public Health Volume 2012, Article ID 312836

²² Jack D. Thrasher PhD sagepub.co.uk/journalsPermission.nav DOI: 10.1177/0748233709348386 tih.sagepub.com

- 4.1.** If we looked at any one of the contaminants in section 2, there are few definitive action levels for any single component. Therefore the health hazard and risk of combined and synergistic effects of multiple toxins and allergens is impossible to assess especially to a population of varying levels of immune response. The absence of measurement cannot be extrapolated to no risk but should be recognised as a current shortfalls in scientific knowledge which cannot assess or even measure risk or hazard which may be presumed to exist. The possible health effects of WDBs can be substantial reflected by the mould *Aspergillus Flavus* which can produce the most toxic natural toxin known to man “Aflatoxin” which does have a maximum exposure level dictated by law.
- 4.2.** The lung tissue, skin or gastrointestinal tract may become inflamed from the recognised allergens always present in WDBs. The absorption of toxins may be presumed to be greater where inflammation (greater blood supply occurs)²³
- 4.3.** The toxic effect of aflatoxin, which is the most toxic natural toxin known to man is classified as a class 1 carcinogen. The toxicity of this agent may be assumed to be increased in the presence of other toxins or allergens, which may slow the bodies particulate mechanical removal system, (ciliary hair beat rate)²⁴, and other toxins known to inhibit the immune system²⁵
- 4.4.** It is recognised that even acute inhalation exposure is at least as toxic as intravenous injection and may be 20 times more toxic than intraperitoneally administered²⁶

5. Crude Risk Assessment

- 5.1.** While some studies²⁷ have shown results from rodent and animal exposure to mycotoxins, none can be extrapolated to humans in the situation described in section 1.3 and the requirement of causation as referred to in “The manual on Scientific Evidence” (Third Edition) is quoted:

5.2. Specific causation or individual causation.

“Established by demonstrating that a defendant’s action or product is the cause of a particular plaintiff’s disease (page 744)²⁸

6. Calculating exposure

²³ Rozman and Klaassen 1996 Morgan et al 1993

²⁴ Coulombe et al 1991 Amitani et al 1995 Sorenson and Simpson 1986

²⁵ Corrier and Norman 1988 Haley 1993

²⁶ Creasia et al 1990 Coulombe et al 1991

²⁷ Z. Islam, et. al., *Satratoxin G from the Black Mold Stachybotrys chartarum Evokes Olfactory Sensory Neuron Loss and Inflammation in the Murine Nose and Brain*, Environmental Health Perspectives, February 27, 2006

²⁸ ISBN 13-978-0-309-21421-6

- 6.1. It is recognised and accepted that the risk of exposure from mould and or associated contaminants in a normal residential or office environments even with limited water or acute water damage can be presumed to be low, however a chronic WDB is not normal and high levels of dose from contaminants is possible.
- 6.2. The European Commission Regulation on aflatoxins from 1999 required that total aflatoxin levels must be less than 4 micrograms/kg in products intended for human consumption⁵. That is more than 100 times higher than what we're permitting in this mathematical model.
- 6.3. We shall assume all the airborne spores and particulates have mycotoxin which are inhaled into the respiratory tract only and discount skin and hand to mouth ingestion.
- 6.4. A list of typical mycotoxins found in WDBs and their "possible" effects can be seen in Table 1.

Mycotoxin	Producing mould	Carcinogenic in humans
Aflatoxin	Penicillium Aspergillus	Liver, lung, hepatitis B
Sterigmatocystin	Penicillium Aspergillus	Liver tumors lung cancer
Ochratoxin	Penicillium Aspergillus	Suspect kidney, pelvis, urethra, bladder,
Zearalenone	Fusarium	Damages chromosomes, kidney and liver, suspected carcinogen and estrogenic (reproductive properties)
Citrinin	Penicillium Aspergillus	Neprototoxic (kidney) mildly hepatotoxic (liver)
Penicillin Acid	Asprgillus	Affects heart
Luteoskyrin	Penicillium	Heptatotoxic and Nephrotoxic carcinogen

Note . Table 1 is shortened from Institute of Medicine Chapter 4 Table 4-5. It must be made clear that not all moulds produce these toxins and dose is a significant factor as is the condition of the immune system of those exposed. The health effects are stated as "known or thought to produced" ²⁹

²⁹ IOM Chapter 4 page 166 Carcinogens

6.5. Table 2 from Jack D. Thrasher PhD Toxicologist and Sandra Crawley
The following list of mycotoxins was taken from a paper which can be
downloaded ³⁰at

Table 2. Mycotoxins produced by toxic molds

Metabolite	Disease	Organisms	Health Concerns
Gliotoxin	Invasive aspergillosis	<i>Aspergillus fumigatus</i> , <i>terres</i> , <i>flavus</i> , <i>niger</i> , <i>Trichoderma virens</i> , <i>Penicillium spp</i> , <i>Candia albican</i>	Immune toxicity, immune suppression, neurotoxicity
Aflatoxin B1; kojic acid; aspergillilic acid; nitropopionic acid	Carcinogenesis	<i>Aspergillus flavus</i>	Liver pathology and cancer; immune toxicity; neurotoxicity
Fumigaclavines; fumitoxins; fumitremorgens; verruculogen; gliotoxin	Aspergillosis	<i>Aspergillus fumigatus</i>	Lung disease; neurotoxicity; tremors; immune toxicity
Ochratoxin A	BEN Urinary tract tumors; Aspergillosis Urinary tract Tumors	<i>Aspergillus niger</i> <i>Penicillium verrucosum</i> <i>Aspergillus ochraceus</i>	Immunosuppression BEN Lung disease Nephropathology
Ochratoxin A Penicillic Acid; Xanthomegnin; Viomellein; Vioxanthin Sterigmatocystin	Carcinogenesis	<i>Aspergillus versicolor</i>	Liver pathology and cancer
5-methoxysterigmatocystin Chaetomiums; Chaetoglobosum A and C	Unknown	<i>Chaetomium globosum</i>	Cytotoxicity Cell division
Griseofulvin; Dechlorogriseofulvins Trichodermin; Trichoderma	Unknown	<i>Memnoniella echinata</i>	Carcinogenesis? Reproductive toxin Hypersensitivity? Protein synthesis inhibition
Mycophenolic acid Botryodiploidin	Unknown	<i>Penicillium brevicompactum</i> <i>Penicillium expansum</i>	Cytotoxic; mutagen Immune toxicity; cytotoxic;
Patulin; citrinin Chaetoglobosin Roquefortine C	Unknown	<i>Penicillium plonicium</i>	Tremors Tremors, cytotoxicity; Nephropathology
Verrucosidins Penicillic acid Nephrotoxic glyco-peptides	Unknown	<i>Trichoderma species</i>	Trichothecene toxicity Immunotoxicity
Trichothecenes Trichodermol Trichodermin Gliotoxin; Viridin Fumonisins	CNS birth defects	<i>Fusarium verticillioides</i> (aka <i>moniliforme</i>) <i>Stachybotrys chartarum</i>	Neural tube defects in animals and humans Respiratory bleeding Protein synthesis inhibition Neurotoxicity Cytotoxicity Immune toxicity
Spirocyclic Drimanes; roridin Satratoxins (F, G, H) Hydroxyroridin E Verrucarins J Trichodermin Dolabellanes Altrones B, C; Stahybotrylactams	Pulmonary bleeding		

³⁰ www.buildingforensics.co.uk resources section (Adverse health effects from mould exposure)

- 1.1. For the purposes of this paper we assume all of the toxin is absorbed by the body, and accumulates over time with none of it being metabolized or broken down. While this is unlikely in a healthy person, risk management dictates that we must assume the persons continuously exposed to mycotoxins over months or years may have depleted health and immune system due to the constant challenges of the WDB.
- 1.2. These and other challenges such as stress and compromised immune systems³¹ may alter NOAEL³² but equally this has not been taken into account. The significant shortfalls of NOAEL as a guideline can be seen from the process of development which is generally derived from in vitro studies which is an artificial environment and these alone cannot be extrapolated into human studies³³
- 1.3. The fungal presence of Water damaged Buildings (WDB) will usually include many spore types, not all of which are sticky slime moulds, such as *Stachybotrys*, which, due to its aerodynamics, are likely to fall quickly although re aerosolized by movement such as walking³⁴ or opening doors.
- 1.4. The size and aerodynamics of a spore, fragment or particulate³⁵ will dictate the time it is a respirable or inhalation risk. Typically a 10micron particle will fall 1 meter in 5 minutes in still air but a 5 micron particle will fall 1 meter in 21 minutes.³⁶
- 1.5. *Penicillium* and *Asprgillus* spores are in the range of 1-2 micron³⁷ and this means they can remain airborne for long periods and be inhaled for deposition lower respiratory areas of alveoli where blood oxygen air exchange occurs³⁸.
- 1.6. The risk of inhalation and exposure to toxins increases as the mould is dried out desiccated as electrostatic charges and the physics of "Brownian Motion" or suspension occurs, reflecting higher dosage in the breathing zone.

³¹ IOM Executive summary page 9

³² No Observed Adverse Effect Levels

³³ IOM Chapter 4 page 126

³⁴ Thatcher and Layton 1995 Ozkaynak et al 1996

³⁵ Gorny et al 2002

³⁶ Hinds 1982

³⁷ Soenson et al 1987

³⁸ Jussila et al 2001

2. The mechanical and toxic effects of Mould and pathogens present in WDBs

- 2.1. The inhalation of particles to the upper respiratory tract may have greater effect from bioavailability than ingestion as spores may lodge in the nasal mucous membranes can damage cells locally or be absorbed into the systemic circulation.³⁹
- 2.2. Many mycotoxins affect residence time and clearance by inhibiting phagocytic activity of macrophages or reducing ciliary beat rate.⁴⁰ The slowing of the body's natural defence mechanisms increases the time for absorption from mould spores, fragments or dust⁴¹
- 2.3. Mycotoxins contaminates present on sub 3 micron particulate may be a greater risk to the lower respiratory areas of alveoli where blood oxygen exchanges occur. This may have greater toxic influence on the body than ingestion of mycotoxin as they may have direct access to blood supply⁴²
- 2.4. The toxic effect of spores and other particulates on alveolar macrophages can impair the ability of these cells to protect against not only mycotoxins but also bacteria and infectious particles⁴³.

3. Calculating dose in the normal building

The following mathematical reasoning is provided from an excerpt of Harriet Berge published paper⁴⁴

- 3.1. One nanogram of mycotoxin ingested is enough to cause an adverse health effect in people. This is extraordinarily conservative although all evidence of mycotoxin (aflatoxin) toxicity to humans has been gathered on ingestion and not inhalation.

³⁹ Morgan et al 1993

⁴⁰ Amitani et al 1995 Coulombe et al 1991 Jakab et al 1994 Sorenson and Simpson 1986 et al

⁴¹ Coulombe et al 1991

⁴² Rozman and Klaassen 1996

⁴³ Coulombe et al 1991

⁴⁴ Harriet A. Burge Health Effects of Biological Contaminates, Indoor air and Human Health. CRC Press 1996 Chapter 10 p171-176

3.2. The European Commission Regulation on aflatoxins from 1999 required that total aflatoxin levels must be less than 4 micrograms/kg in products intended for human consumption⁴⁵. That is more than 100 times higher than what we're permitting in this mathematical model.

3.3. Please note the following published calculations are based on exposure to *Stachybotrys* and not *Aspergillus* (aflatoxin) and lower toxicity levels should be accepted. The level or quantum of toxicity exposure is unknown due to the factors previously discussed.

3.4. A *Stachybotrys* spore is roughly 9.5 x 7.5 µm in size. This is a volume of 2.8 x 10⁻¹⁰ cm³ per spore. Dust with 85% spores has been found to contain 9.5 nanogram (ng) of Satratoxin H (SH)/mg of dust, or 11ng/mg of spores. Note that one ng is just 0.000000001 grams. This yields 3.1x10⁻¹⁵ grams of toxin per spore³.

3.5. A person breathes 30 m³ of air per day and is in this environment all day. This is probably a "reasonably" conservative estimate using data from the California Air Resources Board⁴⁶ and reasonable assumptions about average activity levels throughout the day. We must also accept that exposure can be a combination of inhalation, skin, ingestion or hand to mouth contact⁴⁷

3.6. A background level of 100 spores/m³ of *Stachybotrys* spores. Due to the stickiness of *Stachybotrys* spores, and their relatively fast settling rate, this is a conservative estimate since it would likely require an ongoing active disturbance of a source of *Stachybotrys* to maintain this level continuously or as we have previously stated the presence of other spores or sources of toxin and particulate size.

3.7. Given the model above, it would take over 1,000 days for a person to reach the ten nanogram threshold. The above model provides support for an argument that the risk due to inhalation of fungal mycotoxins in normal office and residential environments is low.

4. Calculating dose or hazard in the WDB

If we accept the calculations presented above as reasonable we should now extrapolate the same calculations to the WDB. In the following tables (appendix) I have listed levels of exposure found in my clients homes over the past 6 months, and significantly these

⁴⁵ <http://www.micotoxinas.com.br/boletim34.pdf> (PDF, 164kb)

⁴⁶ <http://www.arb.ca.gov/research/resnotes/notes/94-11.htm>

⁴⁷ IOM Executive summary page 7

properties had no visible mould and no odour. These statistics are a matter of record and can be substantiated.

4.1. It can be seen from (8.7) that a dose of 100 spores would take over 1000 days to reach the maximum permitted exposure level.

4.2. From the tables of sampling analysis listed in appendix we can see that building occupants were exposed to up to and above 100,000 spores per cubic meter per day and this could reflect in a shock maximum dose in just one day.

5. Overview

5.1. Table 1

This family in Edinburgh lived in a eight year old semi detached property. They requested a survey because they said they had replaced three sets of furniture in 8 years although no mould was visible when I visited the property. The homeowner was blamed for historic mould because she was too clean. During a walk through I saw no visible markers of water damage although I did see shadow on walls and ceiling where mould had previously been painted over by landlords. The whole family had serious if not debilitating respiratory problems. The house was evacuated and tenants re located.

5.2. Overview Table 2

This house was affected by an external flood which entered the house through air bricks below wood floor level. The damage was not mitigated and mould growth from sub floor cavities travelled throughout the property by stack effect. The owners have now become sensitised to the house and are believed to be selling despite decontamination. The property has had a complete and total decontamination and occupants re located for approximately 1 year during works and claim settlement.

5.3. Overview Table 3a 3b 3c

This is a luxury property with absolutely no visible mould. The tenant has aspergillosis and was said to have 3 months to live if he stayed in the property. The property has mould within unknown interstitial cavities and is currently underway with a complete removal of all internal finishes to enable decontamination with an estimated total cost of £1.5million. The family have relocated during decontamination and restoration works estimated to take 12 months.

5.3.1. Table 3a

This table shows the total spore counts in the tens and even hundreds of thousands but note the MEA culture based methods showed very low numbers which is a concern considering these samples were taken at the same time as Total Spore Counts.

5.3.2. Table 3b

This table shows the advantages of aggressive air sampling and the accurate analysis of airborne spores by PCR –DNA and provides economic and detailed

identification and levels of both genus and speciation. This was especially meaningful in this project as there was no visible mould anywhere in the property.

5.3.3. Table 3c

This was the initial sampling undertaken by the landlords “experts” a UKAS accredited laboratory who provide national mould sampling and analysis services. Most importantly their expert report was written and defended by an industry recognised senior industrial hygienist.

Table 3c shows higher ambient mould spores than internally which apart from being wrong was given as an indicator that the property was indeed healthier than outside ambient air. Most importantly the building was said to be safe for the occupant to remain in residence while our subsequent sampling within days of their sampling proved substantial contamination (tables 3a -3b). There is a clear failure of understanding regarding sampling techniques at the very highest level of responsible persons and laboratories which could have resulted in a fatality and has resulted in potential liability issues.

6. Conclusion

- 6.1.** From the tables 1-2-3 and accompanying overview (section 10) it can be seen that visible mould is not an indicator of either mould or associated health hazard and risk.
- 6.2.** Sampling using culture base methodology is recognised in the “WHO Guidelines in Dampness and Mould 2009” as having serious limitations as can be seen in the Tables 1-2-3.
- 6.3.** The levels of mould and associated contaminants so often found in chronic water damaged buildings in the UK can be expected to be a significant risk to immuno compromised individuals which may represent 20% of the population⁴⁸ and serious risk to those recognised as atopic, possibly 40% of the population and a possible allergenic risk to all occupants.
- 6.4.** Expert opinion especially from laboratory analysis using only culture based sampling methods may be suspected as being inaccurate as to possible contamination and health hazards. It is generally accepted that investigation of a WDB should involve a variety of investigation techniques.

I would like to thank the following for their input to this document:

- Jack D. Thrasher Toxicology PhD and Industrial Health 25(9-10) 583–615 The Author(s) 2009 Reprints and permission:
<http://www.sagepub.co.uk/journalsPermission.nav> DOI:
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⁴⁸ Robert Brandy PhD CIH MPH PE CSP CMR

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1. Appendix

1.1. Table 1 Edinburgh

Area	Genus	Total Spore Count
Single bedroom	Penicillium /Aspergillus	58,000
Double bedroom	Penicillium /Aspergillus	44,000
Main Bedroom	Penicillium /Aspergillus	55,000
Lounge	Penicillium /Aspergillus	80,000
Ambient	Penicillium /Aspergillus	170

Table 2 N London

Area	Genus	Total Spore Count
Lounge	Cladosporium	1900
	Penicillium/ Aspergillus	170
Back Room	Cladosporium	3100
	Penicillium/ Aspergillus	1300
Kitchen	Cladosporium	830
	Penicillium/ Aspergillus	93,000
Ambient	Cladosporium	330
	Penicillium/ Aspergillus	170

Table 3a London Total Spore Counts and same time MEA culture base comparison

Area	Genus	Total Spore Count	MEA Culture MPN m ³
Gymnasium	Chaetomium	11,000	>1300
	Cladosporium	6100	>100 cfu
	Penicillium/Aspergillus	120,000	
	Stachybotrys	2600	
Laundry	Cladosporium	1600	>590
	Chaetomium	2000	52 cfu
	Memnoniella	17,000	
	Penicillium /Aspergillus	7600	
	Stachybotrys	5200	
Ambient	Ascospores	5,000	<1300
	Basidiospores	1200	>100 cfu
	Cladosporium	1300	
	Penicillium /Aspergillus	900	

Table 3b PCR DNA Disturbed air Note. Major Genus and stats only listed

AREA	Sample volume m ³	GENUS	Spores per m ³
Gymnasium 1200 litres air	1200	Aspergillus Niger	16,700
		Eurotium	54,000
		Cladosporium	19,000
		Paecilomyces variotii	555,000
		Penicillium Chrysogeuum	112,000
Dinning Room 600 litres air	600	Aspergillus Ochraceus	66,667
		Aspergillus sydowii	<66,667
		Aspergillus ustus	37,000
		Aspergillus versicolor	<6,667
		Eurotium	185,000
		Cladosporium	66,167
		Penicillium chrysogeuum	186,667
		Penicillium pururogenum	<6,667
Penicillium variable	<6,6676		
Ambient	600	Aspergillus sydowii	<3,333

600 litres air	Aspergillus ustus	<333
	Aspergillus versicolor	<333
	Penicillium chrysogenum	<333
	Penicillium pururogenum	<3333

Note on sampling quantity.

The property is a £5 million Grade 11 listed building some two hundred years old. The residence was in pristine decorative condition and no water damage event was recorded. While undertaking a walk through I felt itchy eyes, tingling lips in the gymnasium basement area and this was the focus of my attention and resulted in a larger 1200 litres sampling period to accommodate possible variations.

Table 3c
Culture base analysis 20-25c 120 hours Malt Extract Agar by UKAS laboratory

Area	Genus	CFUs m ³ 200 liters
Basement	Not Known	85
Dining Room	Not Known	150
Ambient	Not Known	275