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1. Introduction

The UK has seen recent discussion regarding the health hazards of WDB (water damaged buildings)¹ and in particular “Toxic mould”

To possibly clear up misinterpretation or doubt the Property Care Association invited an American “expert” Mr Caoimhí P. Connell to present a seminar on mould on 15th November 2011.

Unfortunately many left the seminar believing mould was not a provable health hazard and that any possible risk could not usually be quantified, thereby removing the need for action. Also that measurement of contamination was usually useless as flaws in accuracy made findings un-interpretable.

Many also left believing that recognised internationally and government body accepted decontamination and restoration protocols were based on junk science and that simply drying the building and removing visible mould growth was all that is necessary.

It is my opinion, based on the general conclusions of delegates attending the seminar and opposing reference materials listed in this document, that Mr Connells seminar contained misinformation or information which was not substantiated or provided misleading references.

In my opinion anyone who follows Mr Connell’s advice would be seen to tread a path of folly, contrary to all recognised common duties of care, legislative and legal requirements in the EU and UK and most probably USA too.

¹ IOM Committee Evaluation page 7

While Mr Connell's views may be expressed for discussion they should not perhaps be accepted as "expert opinion" or indeed "fact" and much of his cited evidence or discussion comment is either discredited, seen to be flawed or open for different interpretation as seen in this document.

Equally readers of this draft paper may write substantiated comment in support or refute of its content and my conclusion for inclusion of a supplement final document to be published.

1.1. Summary

The PCA 6 hour seminar was attended by many who had no previous knowledge of mould remediation or general associated health issues and many statements which although intended to alert the delegates to general issues of the "Mould is Gold" industry require review and clarification. This clarification is needed because many of the delegates are stakeholders likely to influence the decision making process of repair to WDBs, where mould and health hazards may affect building occupants. A view generally accepted was that the term the "Toxic mould" was coined by the media reflecting scaremongering, but the term is a shortening of the scientific word "Toxigenic"² and Cytotoxicity³ a recognised risk factor in damage to DNA and RNA associated with some moulds.

Mould toxicity was generally believed to be a relatively new problem which was overblown by the press and the mould remediation industry. The health issues of mould inside buildings has been recognised for several thousand years and reference to health issues and decontamination procedures, (which are similar to today's protocols) can be found in "The Old Testament" Leviticus 13⁴

Part of the evidence presented by Mr Connell was substantiated by references from recognised publications or recognised experts. To avoid the criticism that I have used obscure resources to support my position, I have intentionally used the same reference documents or persons alluded to or quoted at the seminar.

This document may be important due to the fact that many decision makers and stakeholders left the seminar believing that mould is not a serious health hazard or risk. Many believe drying out and removing visible mould is all that's necessary and that sampling or investigation other than visual or olfactory senses was meaningless.

1.2. Reference material

1.1.1. IOM Institute of Medicine Damp and Indoor Spaces (2004)

1.1.2. WHO World Health Organization Indoor Air Quality Dampness and Mould 2009

² ACGIH Chapter 19 page 10 section 19.5.1.1

³ IOM Chapter 2 page 70

⁴ <http://www.enduringword.com/commentaries/0313.htm>

- 1.1.3. Harriet Burge PhD
- 1.1.4. Robert Brandy PhD MPH PE CIH CSP CMR
- 1.1.5. ACGIH American Congress of Governmental Industrial Hygienists
- 1.1.6. AIHA American Industrial Hygiene Association
- 1.1.7. John Hopkins Medicine Health Alerts (section 8)
- 1.1.8. HSE Health & Safety Executive

2. General Overview

2.1. The general health hazards of water damaged buildings and risk based appraisal.

2.2. For the purposes of clarification water damaged building includes⁵:

- 2.2.1. Flooding
- 2.2.2. Dampness,
- 2.2.3. Condensation
- 2.2.4. High relative humidity,
- 2.2.5. Water ponding
- 2.2.6. Microbial growth

It is accepted mycotoxins are sometimes released as a defence against challenges by other moulds or bacteria or indeed when under attack by desiccation or application of fungicide.

2.3. Fungi Spore casing or outer shell contain known and potentially allergenic⁶ components such as β 1-3 verified by antigen characterisation⁷ Mycelium also release sub micron potentially allergenic particulates considered capable of penetrating deep into the alveolar⁸

2.4. It is accepted that not all water damaged homes become a health hazard especially where prompt action prevents bio amplification or where environmental conditions are not commensurate for growth.⁹

2.5. There is therefore, a potential for the recognised health hazards from mould and associated contaminants in all water damaged buildings especially where environmental conditions can alter to provide ideal growth conditions over time or where dose related issues are extrapolated over time.¹⁰

2.6. The Chronic effects of water damage and associated possible health issues may be caused by:

2.6.1. Condensation through fuel poverty

⁵ IOM Executive summary, Committee Evaluation page 7

⁶ IOM Chapter 2 page 67

⁷ IOM Chapter 2 page 66

⁸ IOM Chapter 2 page 66

⁹ WHO Chapter 5 page 93

¹⁰ IOM Chapter 1 page 24

- 2.6.2.** Leaks, penetrating, or rising damp
 - 2.6.3.** Poor ventilation
 - 2.6.4.** Poor remediation of a flood damaged building resulting in historic and continuing exposure from usually “non viable” (dead) mould spores after the building has dried naturally.
 - 2.6.5.** Poor insulation (“Dew Point” condensation)
- 3.** The health hazards most often associated with water damaged buildings and their related agents are respiratory¹¹ but the following health complaints are also identified by the Institute of Medicine as potential risks, which may be circumstantial due to the lack of extensive research¹² It should be recognised that absence of evidence is not evidence of absence and complex hazards and risks can be present and assessed by competent investigation.

3.1. Skin¹³

- 3.1.1.** Eczema
- 3.1.2.** Atopic dermatitis

3.2. Gastrointestinal¹⁴

- 3.2.1.** Vomiting
- 3.2.2.** Diarrhea

3.3. Fatigue and Neuropsychiatric¹⁵

- 3.3.1.** Unusual tiredness
- 3.3.2.** Lethargy
- 3.3.3.** Concentration
- 3.3.4.** Depression
- 3.3.5.** Headache¹⁶

3.4. Cancers¹⁷

- 3.4.1.** Several “common” fungi produce mycotoxins which are carcinogenic, and inhalation can result in the production cytotoxic agents.¹⁸

3.5. Reproductive issues¹⁹

- 3.5.1.** Challenging because of multiple factors and lack of research

3.6. Rheumatic and other immune diseases²⁰

3.7. Inflammation and or stiffness or pain in muscles, joints

¹¹ IOM Chapter 5 page 243

¹² IOM Chapter 5

¹³ IOM Chapter 5 page 244

¹⁴ IOM Chapter 5 page 245

¹⁵ IOM Chapter 5 page 247

¹⁶ IOM Chapter 5 page 250

¹⁷ IOM Chapter 5 page 250

¹⁸ IOM Chapter 5 page 251

¹⁹ IOM Chapter 5 page 251

²⁰ IOM Chap[ter 5 page 251

3.8. Exacerbation of above in damp homes.

3.9. Suppression of Immune system²¹

3.10. Cardiovascular²²

3.10.1. Tachycardia

3.10.2. Low blood pressure²³

3.11. University of Reading and Royal Institute of Chartered Surveyors Foundation²⁴

3.11.1. Illness can result from high level short term exposure or low levels of mould for long term exposure

3.11.2. International review of literature, "Mould plays a major role in exacerbation of Asthma and allergies

3.11.3. The mould problem is being compared to the 'asbestos experience' two decades ago. However the problem is further complicated by its biological rather than chemical origins. Mould has the ability to grow and transform, which makes laboratory samples and site investigations far more complex.

3.11.4. Claims from the workers and the users of affected buildings are likely to become more prevalent because of the rapidly growing publicity, hype and litigation, involving allegations of mould damage. A number of states in the USA are involved in legal proceedings. The insurance company General Cologne Re (*Kingdollar, 2001*) reports that several toxic mould awards and/or settlements have exceeded US\$500,000 in damages.

3.11.5. The most common symptoms reported from exposures in indoor environments are runny nose, eye irritation, cough, congestion, aggravation of asthma, headache, and fatigue.

3.11.6. In order for humans to be exposed indoors, fungal spores, fragments, or metabolites must be released into the air and inhaled, physically contacted (dermal exposure), or ingested. Whether or not symptoms develop in people exposed to fungi depends on the nature of the fungal material (e.g., allergenic, toxic, or infectious), the amount of exposure, and the susceptibility of exposed persons. Susceptibility varies with the genetic predisposition (e.g., allergic reactions do not always occur in all individuals), age, state of health, and concurrent exposures. For these reasons, and because measurements of exposure are not standardized and biological markers of exposure to fungi are largely unknown, it is not possible to determine "safe" or "unsafe" levels of exposure for people in general.

3.11.7. Immunological reactions include asthma, and allergic rhinitis. Contact with fungi may also lead to dermatitis. It is thought that these conditions are caused by an immune response to fungal agents. The most common symptoms

²¹ IOM Chapter 4 page 125

²² ACGIH 24.2.2.4

²³ ACGIH 24.2.2.4

²⁴ University of Reading The risk of mould damage over the whole life of a building 2003 (RICS Foundation)

associated with allergic reactions are runny nose, eye irritation, cough, congestion, and aggravation of asthma

3.11.8. A wide variety of symptoms have been attributed to the toxic effects of fungi. Symptoms, such as fatigue, nausea, and headaches, and respiratory and eye irritation have been reported. Some of the symptoms related to fungal exposure are non-specific, such as discomfort, inability to concentrate, and fatigue.

3.11.9. Severe illnesses such as Organic Dust Toxic Syndrome (ODTS) and pulmonary hemosiderosis have also been attributed to fungal exposures. ODTS has been documented in farm workers handling contaminated material, but is also of concern to workers performing renovation work on building materials contaminated with fungi.

3.12. Mr Connell further went on to say that “Dose makes the poison “ and that the presence of even extremely toxic chemicals was not a risk if they were contained or at such a low level as to be an insignificant risk. It was suggested that instead of stating the toxin present, prove the dose by measurement and comparison against permitted exposure levels.

3.13. Discussion and the Law Note JC (Jeff Charlton)

There is a requirement in EU and UK law to protect employees from exposure to hazardous substances²⁵ and as mycotoxins are the most toxic natural substance known to man and they are used as reference point against other toxins²⁶. There is an obvious risk of mycotoxins and other contaminants in a water damaged building, a point accepted by CIRIA²⁷ and of course by the usual process of risk and hazard assessment which the HSE²⁸ provide guidance as “Five steps to Risk Assessment”²⁹

3.14. Risk and hazards are described by the HSE as:

3.14.1. **Hazard** is anything that may cause harm, such as chemicals, electricity, working from ladders, an open drawer, etc;

3.14.2. **Risk** is the chance, high or low, that somebody could be harmed by these and other hazards, together with an indication of how serious the harm could be.

3.15. The law requires compliance to these issues and failure to comply under section 37 HSW Act 1974 can result in fines and imprisonment³⁰

3.16. While the forgoing reflects the employers duties to their employees it should be recognised that workers are usually healthy and engaged in work on a water damaged property for less than 8 hours per day. A standard duty of care must be

²⁵ Control of Substances Hazardous to Health 2002

²⁶ ACGIH Chapter 24.2.2.3

²⁷ <http://www.ciria.com/flooding/disinfection.htm>

²⁸ Health and Safety Executive

²⁹ <http://www.hse.gov.uk/risk/fivesteps.htm>

³⁰ <http://www.hse.gov.uk/leadership/legislation.htm>

recognised to the building occupant often of unknown health status and possibly exposed to the same hazards and risk as employees for 24/7.

3.17. The hazards of a water damaged building include toxic chemicals, allergens, and irritants. Typical exposure concerns are stated on the HSE “Advisory Committee on Dangerous Pathogens” where water damage type hazards are listed³¹

3.18. The presence of water damage related hazards can be in the form settled dust or aerosolised particulates, with exposure routes of skin or ingestion and inhalation of bioaerosols all of which can present a recognised health hazard as described in HSE document on poultry dust³² and in particular provides guidance for the application of COSHH for allergenic “dust” substances.

3.19. Most significantly this paper identifies serious health risks from many of the constituents present in a water damaged buildings, typically moulds such as Cladosporium, Aspergillus, Fusarium, together with risks of endotoxins and bacteria,³³ volatile organic chemicals and reference is made to EH40 which is a list of workplace exposure levels of some known and recognised chemicals³⁴.

3.19.1. JC (Jeff Charlton) Note

The list of approximately 600 recognised hazardous chemicals in the USA was published in the 1960s. The list in the UK is over 3000 but new chemicals are being introduced at a rate of several hundred each week making regulation or guidance almost impossible.

3.20. Exposure levels are another significant point but should be recognised as levels of single point exposure.

3.21. In EH40³⁵ we see that Benzene has a TWA³⁶ of Benzene 1ppm as a maximum permitted exposure level we might assume to be safe, but if we then added Bromine .1 and then added Calcium Hydroxide, and a hundred other contaminates the challenge to the human body may overwhelm its defence mechanisms.

This is exactly what happens in a water damaged building scenario, where many different potentially pathogenic materials are uncontrollably released not for 8 hours, but possibly exposing building occupants for months and years. Therefore the compound hazards and associated risks of unknown unquantifiable components of a water damaged building must be seen as a serious issue for risk managers.

3.22. Standard operating protocols for unknown hazards is engineering controls such as:

3.22.1. Avoidance

3.22.2. Control

3.22.3. Removal

³¹ <http://www.hse.gov.uk/pubns/misc208.pdf>

³² <http://www.hse.gov.uk/pubns/web40.pdf>

³³ <http://www.hse.gov.uk/pubns/web40.pdf>

³⁴ <http://www.hse.gov.uk/cosHH/table1.pdf>

³⁵ <http://www.hse.gov.uk/cosHH/table1.pdf>

³⁶ Time Weighted Average over 8 hours PPM

3.22.4. Dilution

3.22.5. Neutralisation

3.22.6. PPE is recognised as a last option after or to supplement engineering controls.

3.23. Probably the most significant reference in this review is a paper published by the HSE which identifies the hazards and risk elements of a composting³⁷.

3.24. It should be recognised that a water damaged building is in decay from the same chemical and biological actions involved in composting or natural decay of carbohydrates and cellulose materials and therefore in my opinion, absolutely no doubt exists as to potential hazards likely to be present in a WDB and specifically the very high risk to some members of the general population due to long term exposure of low dose or short term exposure of high levels of toxin.

3.25. With the potential toxic compound effect of many unknown and possibly yet to be identified genus of mould being present in a water damaged building, coupled to chemicals expected to be present, the hazard is usually impossible to quantify.

3.26. The quantification of the unknown hazard is therefore impossible although some realistic markers such as mould rank order may provide guidance and this may be coupled to other quantitative or qualitative techniques.

3.27. While measurement of the unknown, often invisible hazard may not be economically possible, its presence or possible absence cannot be ignored or taken for granted. Following normally accepted basis for risk assessment it would be seen as prudent to assume the presence of a hazard where evidence to the contrary is absent especially where evidence of potential risk exists.

3.28. Risk assessments should, in my opinion, be based on severity and time which would likely result in the bio amplification of hazards subject to environmental conditions, growth media present and coupled to assessments of risk groups exposed. Risk assessments can and should in my opinion utilise any suitable, economic realistic sampling or investigation principles which provide substantiation of risk hypothesis.

3.29. The example of USA risk management presented at the PCA seminar, is that if you are unable to establish dose before adverse health effects occur from an exposure, then it can be concluded, the exposure is not a health risk worth taking precaution.

3.29.1. This opinion has successfully been challenged in USA courts based on requirements of scientific evidence³⁸ and similarly in the UK or EU absence of evidence is not evidence of absence especially when many of the chemicals likely to be present are recognised in COSHH and other reference materials.

3.30. Reference Manual on Scientific Evidence (Third edition)³⁹

3.30.1. Specific causation or individual causation.

³⁷ <http://www.hse.gov.uk/research/rrpdf/rr130.pdf>

³⁸ <http://freepdfhosting.com/564816a98e.pdf>

³⁹ ISBN 13-978-0-309-21421-6

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

4. Duty to undertake risk assessment of toxic substances⁴⁰

The following guidance highlights procedures and duties to protect employees. It should of course be recognised that employees are often exposed during an 8 hour working day for a typical 40 hour working week. Building occupants, especially residents however may be exposed 24/7 for years. There is a legal duty to protect employees and this may be seen to be extrapolated to cover other occupants of a workplace (home) from a duty of care perspective.

4.1. The Control of Substances Hazardous to Health Regulations 2002 (as amended) (COSHH),¹ regulation 2, states that substances and preparations (mixtures of two or more substances) hazardous to health include⁴¹:

4.1.1. substances in Part 1 of the *Approved supply list 2* as dangerous for supply within the meaning of the Chemicals (Hazard Information and Packaging for Supply) Regulations 2002 (CHIP)³ and for which an indication of danger specified for the substance is very toxic, toxic, harmful, corrosive or irritant

4.1.2. Biological agents;

4.1.3. Any substance that creates a risk to health because of chemical or toxicological properties.

4.2. The principal regulations governing substances hazardous to health are the COSHH Regulations. The central requirements are⁴²:

4.2.1. Regulation 6(1) - you should carry out a suitable and sufficient assessment of the risks to the health of your employees and any other person who may be affected by your work, if they are exposed to substances hazardous to health;

4.2.2. Regulation 7(1) - you should ensure that exposure is prevented or, when this is not reasonably practicable, adequately controlled.

4.3. The legal requirement for monitoring inhalation exposure is given in regulation 10 of the COSHH Regulations. It requires duty holders to carry out monitoring if.⁴³

4.3.1. it is not immediately obvious to you whether there is a risk to the health of your employees; and

4.3.2. there is a suitable procedure which you can use to measure exposure.

⁴⁰ HSE Monitoring strategies for toxic substances 2006

⁴¹ HSE Monitoring strategies for toxic substances 2006 section 5 page 2

⁴² HSE Monitoring strategies for toxic substances 2006 section 10 page 5

⁴³ HSE Monitoring strategies for toxic substances 2006 section Legislative requirements page 5

- 4.4. Regulation 10 is clarified in detail in the COSHH *Approved Code of Practice*.⁶ This states that monitoring is required when:
- 4.4.1. failure or deterioration of the control measures could result in a serious health effect;
 - 4.4.2. when measurement is needed to ensure a WEL⁴⁴ or any self-imposed (in-house) working standard is not exceeded; or
 - 4.4.3. as an additional check on the effectiveness of any control measures provided in accordance with regulation 7, and always in the case of the substances or processes specified in Schedule 5 to the Regulations;
 - 4.4.4. when any change occurs in the conditions affecting employees' exposure which could mean that adequate control is no longer being maintained.
- 4.5. To comply with the requirements in COSHH regulation 7(7)(c) (exposure to asthmagens and carcinogens) to reduce exposure so far as is reasonably practicable, employers may need to carry out a programme of air monitoring in accordance with regulation 10. This will be generally necessary unless the risk assessment shows the exposure is unlikely to ever exceed the WEL.⁴⁵
- 4.6. The majority of substances used in industry have not been given WELs, but this does not mean that they are safe. In these circumstances you need to ensure that exposure is controlled to a level to which nearly all the working population could be exposed day after day, without adverse effects on their health.

Editors Note. *The Atopic population could be between 5 and 40% and dose may affect the non atopic population too*⁴⁶

- 4.7. To judge whether there is a risk to the health of your employees in these circumstances, you may be able to use limits produced by other bodies, such as *Guide to occupational exposure values 2005* from the American Conference of Governmental Industrial Hygienists. ***In all cases, the principles of good control practice should be applied in the first instance.***⁴⁷
- 4.8. **Initial Appraisal**⁴⁸The initial appraisal is an important part of the strategy described in Figure 2. It helps you establish the need for, and the extent of, exposure monitoring. This will help you to decide on:

- 4.8.1. The hazards;
- 4.8.2. The potential risks; and

⁴⁴ Worker Exposure Limit

⁴⁵ HSE Monitoring strategies for toxic substances 2006 section Legislative requirements page 6

⁴⁶ ACGIH Chapter 25 .1

⁴⁷ HSE Monitoring strategies for toxic substances 2006 section Exposure Limits page 6

⁴⁸ HSE Monitoring strategies for toxic substances 2006 section Initial Exposure page 10

4.8.3. Whether more information is needed (eg do you need to carry out monitoring to obtain an estimate of the exposure of your employees to substances hazardous to health?).

4.9. The first stage of the initial appraisal requires you to find out information on a variety of factors, for example⁴⁹:

4.9.1. the substance that your employees are exposed to;

4.9.2. the hazardous and physical properties of the substance;

4.9.3. the airborne forms of the substance;

4.9.4. the processes or operations where exposures are likely to occur;

4.9.5. the number, type and position of the sources from which the substance may be released;

4.9.6. which groups of employees are most likely to be exposed;

4.9.7. the pattern and duration of exposure;

4.9.8. work practices;

4.9.9. the means by which the release of the substance is controlled;

4.9.10. whether respiratory protective equipment and/or other personal protective equipment are worn and their effectiveness; and

4.9.11. what are the WELs, limits?

4.10. Various qualitative methods of tracing the route of contamination can be used including smoke tubes, particle counters but although smell can be an indicator of contamination it is an unreliable method.⁵⁰

4.11. Alternatively, validated laboratory-based sampling and analytical techniques can be used (see Appendix 2). If you are unsure how to use such techniques, you could consult a health and safety professional such as an occupational hygienist⁵¹.

4.12. The various risk assessment methods listed in the document are designed to provide an initial view as to the possible presence of hazard, extent of possible risk and the possible requirement of quantitative assessments or initiation of a process to control exposure⁵²

4.12.1. The approach can be used when:

the extent and pattern of exposure cannot be confidently assessed by a basic survey;

4.12.1.1. exposure is highly variable between employees doing similar tasks;

4.12.1.2. carcinogenic substances (risk phrase R45 and R49),

4.12.1.3. respiratory sensitisers (risk phrase R42 and R42/43) or mutagens (risk phrase R46) are involved;

4.12.1.4. the initial appraisal and basic survey suggest that:

⁴⁹ HSE Monitoring strategies for toxic substances 2006 section Initial Exposure page 10

⁵⁰ HSE Monitoring strategies for toxic substances 2006 section Initial Exposure page 11

⁵¹ HSE Monitoring strategies for toxic substances 2006 section Initial Exposure page 12

⁵² HSE Monitoring strategies for toxic substances 2006 section Initial Exposure page 12

4.12.1.5. the time-weighted personal exposure may be very close to the WEL, limits from another body or in-house standard; and

4.12.1.6. - the cost of additional control measures cannot be justified without evidence of the extent of exposure variability;

4.13. Significant points of reference

4.13.1. you must reduce exposure to the point where there is a big difference between, on the one hand, the sacrifice (in money, time or trouble) that would be involved in further measures and, on the other hand, the risks from exposure (which should be insignificant)⁵³.

4.13.2. physical and chemical properties of the substance such as the vapour pressure, boiling point and particle size;⁵⁴

4.13.2.1. **JC note.** VOCs, MVOCs, chemical toxins, allergens are present in mould and WDB

4.13.3. the number of sources from which the substance is released;

4.13.3.1. **JC note.** Hidden sources in cavities should be considered as potential routes of exposure

4.13.4. ambient conditions (temperature, pressure and humidity).⁵⁵

4.13.4.1. **JC note.** These issues are a major factor in bioamplification and increased risk

4.13.5. the length of time the employee spends in the vicinity of the source⁵⁶;

4.13.5.1. **JC note.** Employees are usually at work for 8 hours per day but building occupants may be exposed 24/7 and be unhealthy or have depressed immune systems thereby being at much greater risk from exposure.

4.14. Health Effects

4.14.1. The type of health effect that could be induced by the substance will influence the monitoring procedure. For a substance with acute effects such as eye irritation, the monitoring techniques need to be sensitive enough so that peak and short-term (15-minute TWA) measurements can be taken.

4.14.2. In the case of chronic effects the monitoring strategy will mainly focus on long-term (eight-hour TWA) exposures. This is because, in general, the development of health effects depends on exposure over a prolonged period of time.

⁵³ HSE Monitoring strategies for toxic substances 2006 Compliance testing page 15

⁵⁴ HSE Monitoring strategies for toxic substances 2006 Compliance testing page 16

⁵⁵ HSE Monitoring strategies for toxic substances 2006 Compliance testing page 16

⁵⁶ HSE Monitoring strategies for toxic substances 2006 Compliance testing page 16

4.14.3. 4 You may need to consider substances which have the potential to induce both acute and chronic effects. The monitoring strategy will need to take this into account.

5. Mould sampling

Sampling is a small part of an investigation into a property where occupant health is a concern and usually follows a chronic or long standing water damage event. The sampling strategy where or if necessary should follow a walk through of the building to assess visible markers, coupled to other assessments such as known or suspected health effects of occupants, and developed to prove a hypothesis of cause and effect⁵⁷.

This is because mould requires suitable environmental factors for growth although mould may only be a single component of contamination it is often a useful indicator of other issues being present.⁵⁸

Sampling should not be undertaken routinely and usually where mould is visible it is almost always unnecessary unless the medical profession need to know genus and speciation⁵⁹. Where sampling is undertaken it should be seen as part of an overall risk assessment and lab analysis and results should be viewed as indicators of a snap shot in time of circumstances at the point of sampling.

5.1. Typically sampling decisions should include but not be limited to the following factors⁶⁰

- 5.1.1.** Specific disease or symptoms
- 5.1.2.** Acute v Chronic health outcomes
- 5.1.3.** Population v patient base approach
- 5.1.4.** Suspected exposure variation
- 5.1.5.** Available methods to measure individual agents
- 5.1.6.** Cost of sampling

5.2. A significant factor in sampling quantification is the use of information gained, whether you have 2000 spores per cubic meter or 5,000 the actions are likely to be the same; however rank order⁶¹ is internationally seen as a guide to the building condition in terms of contamination. Mould sampling can sometimes be the only guide to identifying a health hazard especially if the building has been repaired, dried and decorated and the only known issue is the health complaints or risk of the occupants.

5.3. Sampling techniques recognised⁶² should be designed to identify the specific or general complex mixture of (live (viable) and dead (non viable) fragments, toxins,

⁵⁷ ACGIH Chapter 4

⁵⁸ ACGIH 19.5.1.1 and 24.5.2 section 24 generally

⁵⁹ IOM chapter 5

⁶⁰ IOM Chapter 4 page 95

⁶¹ IOM Chapter 2 Page 59

⁶² IOM Chapter 3 page 90

allergens, MVOCS⁶³ It should be noted that many traditional sampling techniques such as culture base (SAS) air sampling and settle plates provide extremely limited information against the new techniques available today⁶⁴.

5.4. Sampling may be of use where health concerns or potential risks of contamination exist but where visible markers of mould or water damage are absent. This may be due to previous poor remediation or redecoration. Mould hidden in cavities can move into the breathing zone of building occupants and therefore cavity sampling can sometimes be justified⁶⁵.and ⁶⁶

5.5. The following issues are raised regarding the accuracy and usefulness of sampling:

5.5.1. Sampling isn't accurate

Sampling is not accurate because it is a reflection of circumstances at that time and environmental conditions can mean different results may be gathered in the same place even second later. The accuracy of sampling can greatly be increased by more sampling⁶⁷ due to the episodic nature of contamination levels in a dynamic environment,⁶⁸ but this can increase costs out of proportion to usefulness and usefulness should not be confused with perfection.⁶⁹The added cost of sampling to provide more accurate results may not be justified especially when other historic records of similar buildings is available⁷⁰.

5.5.2. All sampling has shortfall and benefit but some techniques are seen as providing limited information such as culture base to the more recognised Total Spore Count and more recently the Mould Specific PCR technology which is recognised as providing more meaningful results⁷¹ As many contamination movement events are episodic accuracy can be extremely time consuming and expensive and costs must be assessed together with result benefit.

5.5.3. Lab results vary⁷²

This is unfortunately true and it is therefore essential that only labs are used which can prove a recognised quality control program⁷³ These sampling problems and

⁶³ Microbial Volatile Organic Chemicals

⁶⁴ WHO Dampness and Mould Guidelines 2009 Chapter 2

⁶⁵ IOM Chapter 6 page pages 289-290-291-292-300

⁶⁶ ACGIH Chapter 2

⁶⁷ IOM Chapter 3

⁶⁸ IOM Chapter 3 page 98

⁶⁹ Written comment from Harriet Burge received by Jeff Charlton

⁷⁰ IOM Chapter 3 page 99

⁷¹ WHO Chapter 2

⁷² ACGIH Chapter 6 6.9.2

⁷³ R Brandy

adjustment are not unique to mould sampling. Similar problems occur with sampling other particulates and chemicals⁷⁴

5.5.4. Sampling provides no useful information

If a walk through survey could not identify typical visual markers hidden mould or water damage may be missed. Markers may be hidden or camouflaged by redecoration or poor remediation practice. Sampling can assist in identifying the hidden issues and more importantly provide comparisons between what can be assessed as normal or elevated.

5.5.5. Mould sampling can identify rot, genus that do not grow indoors and indicate comparisons between normal flora often seen as ambient and rank order differences indicating or confirming the presence of mould and or water damage.⁷⁵

Sampling in water damaged buildings should reflect the required outcome and many different testing protocols are available all of which provide different information depending on what is required. Some moulds present inside a building require urgent action and controlled “safe” removal⁷⁶

5.5.6. Sampling not necessary for cavities?

The presence of visible mould, even toxigenic⁷⁷ strains is not an absolute indicator that health problems exist, equally the absence of visible mould is not an indicator that the building is free from contamination in hidden sources.⁷⁸ Various bodies including the EPA and AIHA discuss practical cavity investigation with specific caution on spore release.⁷⁹

The potential for mould exposure from wall cavities, attics, sub floor is poorly understood⁸⁰ and therefore must rely upon sampling or risk assessment. The size of observed water or mould damage has some relevance to possible severity of exposure however it should be remembered that damage is often hidden⁸¹ and therefore either intrusive investigation or air sampling may be required.

5.5.7. Sampling not necessary for clearance

In the majority of straight forward water damaged buildings where small amounts of mould are isolated to an adjacent and recognised water loss, or time lines to drying have been adequate, simple cleaning to remove visible mould may be all that is necessary and clearance should not be necessary. In long term or chronic events however the contamination may be both visible and invisible and hazards and resulted risks unknown or invisible.

⁷⁴ R Brandy

⁷⁵ IOM Chapter 2 page 59-64

⁷⁶ ACGIH Chapter 19

⁷⁷ IOM Chapter 2 page 70

⁷⁸ IOM Chapter 7 page 317

⁷⁹ IOM Chapter 6 page 277

⁸⁰ IOM Executive summary page 12

⁸¹ IOM Chapter 2 page 50

5.5.8. The contaminants mentioned throughout this document are often invisible, some are confirmed as toxic such as Aspergillus and if you didn't undertake air sampling how could you confirm you have located all sources of contamination.

While many contractors are competent, many are not and the use of a third party to provide some evidence of protocol compliance and clearance, occupants safety and health may be put at risk. There are several international standards, (none of which are American) for mould clearance⁸² or recognised acceptability.

6. Health effects

The health hazards of mould and other associated pathogens present in water damaged buildings are internationally recognised⁸³

- 6.1.** Some moulds found to grow in water damaged buildings can produce the most potent natural carcinogen known to man and this is used as a benchmark measure against the carcinogenicity of other chemicals.⁸⁴ Exposure can be by inhalation and ingestion⁸⁵ and also skin contact.
- 6.2.** Heart conditions such as Tachycardia, low blood pressure, and gangrene can be caused by mycotoxin exposure⁸⁶
- 6.3.** Moulds and bacteria sometimes present in water damaged buildings produce molecules known or thought to cause cancer in humans and other animals.⁸⁷ Dose or exposure required to cause toxic effects has yet to be determined⁸⁸
- 6.4.** The complex nature of synergistic effects of multiple contaminants makes the identification of sole causation extremely difficult when assessed against the differing response of the atopic and allergenic population⁸⁹ While the population groups known as sensitised or susceptible are recognised as Atopic or allergenic, it is recognised that nonatopic suffer adverse health effects in water damaged buildings too⁹⁰.
- 6.5.** As the health effects of water damaged buildings cannot be quantified⁹¹ Temporal bio amplification is a recognised issue and effective fast control and remediation of

⁸² R. Brandy PhD CIH Worldwide exposure standards.

⁸³ ACGIH 24.5.2 and 19.5.1.1

⁸⁴ ACGIH Chapter 24.2.2.3

⁸⁵ ACGIH 24.2.2.3

⁸⁶ ACGIH 24.2.2.4

⁸⁷ IOM Chapter 4 page 166

⁸⁸ Chapter 4 page 170

⁸⁹ WHO Chapter 2

⁹⁰ WHO Executive summary X111

⁹¹ WHO Executive summary XV

water damaged buildings is essential to reduce human exposure to possibly present pathogens⁹².which may be expected to amplify over time.⁹³

- 6.6.** Apart from mould other pathogens such as endotoxins, bacteria, virus and volatile organic chemicals are likely to affect building occupants health either in terms of chronic or acute exposure⁹⁴ and ⁹⁵
- 6.7.** Mould is often seen as an indicator of other contaminants likely to be present which may affect both atopic and non atopic population⁹⁶. In terms of health there is little doubt that people who live in water damaged or damp buildings are more likely to have poor health⁹⁷.These health risks are increased in poorly maintained low income households who may at increased levels of risk⁹⁸ possibly due to fuel poverty or unresolved building defects.
- 6.8.** The issue is “Absence of Evidence is not Evidence of Absence and from data produced by the WHO and IOM, and ACGIH a substantial risk of health hazard can be seen to possibly exist and this must be assessed⁹⁹. The risk and hazard assessments required by duty holders must reflect the recognised multiple hazards often present in some water damaged buildings.¹⁰⁰ The risks from mould exposure is accepted for workers who generally should wear full face respirators fitted with ABECK cartridges¹⁰¹ and utilise engineering controls.
- 6.9.** CIRIA ¹⁰² the building research and information service have identified the health hazard and risk of mould after just three days of building materials being wet¹⁰³.
- CIRIA recommend sensitised people and contractors engaged in water damage restoration should wear well fitting respirators fitted with toxic particle cartridges and this guidance is backed sponsored by the ABI.¹⁰⁴
- 6.10.** High exposure from spore release can occur within 7 days of a water loss event and within one day of moist conditions.
- 6.11.** The substrate or building material mould develops on, coupled to challenges from other mould or bacteria may influence its toxicity¹⁰⁵.

⁹² WHO Chapter 2

⁹³ WHO

⁹⁴ WHO Executive summary XIV

⁹⁵ ACGIH Chapter 24

⁹⁶ IOM Chapter 4

⁹⁷ WHO Executive summary X111

⁹⁸ Who Executive summary XV

⁹⁹ ACGIH 24.2.2.3

¹⁰⁰ HSW Act 1974 and COSHH 2002 and MHSW Regs 1999 also HHSRS 2004

¹⁰¹ IOM Chapter 6 page 280-281

¹⁰² Construction Industry Research and Information Service

¹⁰³ <http://www.ciria.com/flooding/disinfection.htm>

¹⁰⁴ Association of British Insurers, sponsorship of “Preparing for Floods”

¹⁰⁵ IOM Chapter m2 page 70

- 6.12.** The Health and Safety Executive and BISRIA recognise the health hazards of mould and the risk to health¹⁰⁶

7. Decontamination

Various standards or guidelines regarding decontamination and clearance exist¹⁰⁷ and these generally revolve around the US Government EPA information¹⁰⁸ standards often recognised as NYCG¹⁰⁹ and the following US and UK industry and government accepted guidelines¹¹⁰

7.1. Trade Associations

- 7.1.1.** Institute for Inspection, Cleaning and Restoration Certification (IICRC) S500 (2006) 8
- 7.1.2.** Institute for Inspection, Cleaning and Restoration Certification (IICRC) S520 (2003) 9
- 7.1.3.** American Industrial Hygiene Association (AIHA) 2004 Document 10
- 7.1.4.** American Conference of Governmental Industrial Hygienists (ACGIH) 1999 Document 11
- 7.1.5.** National Air Duct Cleaners Association (NADCA) ACR 2001, 2006 12
- 7.1.6.** Association for the Prevention and Study of Contamination (ASPEC) 2004 13
- 7.1.7.** Institute for Research on Occupational Health and Safety (IRRST) 2004 13
- 7.1.8.** International Society of Indoor Air Quality and Climate Guidelines (ISIAQ) (1996)
- 7.1.9.** UK Indoor air quality and mould in ventilation systems.¹¹¹

7.2. Governmental Bodies

- 7.2.1.** Federal Environmental Protection Agency (USEPA) 2001 Guideline 14
- 7.2.2.** UK Ministry of Defence 2004- Requirements for air-conditioning and ventilation
- 7.2.3.** New York City Department of Health (NYCDOH) 2002 Document 16
- 7.2.4.** Texas Department of Health (TDH) 2004 Regulation 17
- 7.2.5.** Occupational Safety and Health Administration (OSHA) 2003 Document 18
- 7.2.6.** University of Minnesota Bulk Analysis of Interior Ventilation Duct Insulation 19
- 7.2.7.** USACE / NAVFAC / AFCESA / NASA-Unified Facilities Guide Specifications

- 7.3.** These general protocols often assess visible mould growth in general terms of square feet and levels of potential exposure. In simple terms HEPA sandwich or vac, damp wipe and vac using a HEPA rated vacuum cleaner is sometimes sufficient in a newly contaminated property with minor mould contamination, however the success of this technique is limited as can be seen from UK¹¹² and US¹¹³ decontamination anthrax events where detailed analysis proved significant failures in the HEPA sandwich process. This simplistic view does not take account of larger than 10 square feet of contamination, or active mould growth on soft or porous materials.

¹⁰⁶ HSE <http://www.hse.gov.uk/research/rrpdf/rr471.pdf> Chapter 4 page 6

¹⁰⁷ World Wide exposure standards for mould and bacteria R Brandy PhD CIH

¹⁰⁸ <http://www.cdc.gov/mold/cleanup.htm>

¹⁰⁹ <http://www.nyc.gov/html/doh/html/epi/moldrpt1.shtml>

¹¹⁰ http://www.certifiedcleaners.org/pr_ANSI-Approved-S520.shtml

¹¹¹ HVCA TR19

¹¹² Scottish farmhouse and village hall contaminated with natural anthrax 2007

¹¹³ Brentwood postal facility anthrax event

Most decontamination works can reasonably be undertaken by competent contractors.

7.4. The effectiveness of this type HEPA sandwich is in some doubt regarding the air quality as extensive surface cleaning is unlikely to reduce airborne contamination sufficiently in heavily contaminated buildings.¹¹⁴ Decontamination protocols should be designed to protect both the restoration contractor and building occupant¹¹⁵

7.5. Engineering controls should be seen as a first line of protection and PPE as a secondary but essential requirement¹¹⁶.

7.6. A review of various approaches to mould remediation exist¹¹⁷ and these generally require containment and engineering controls for anything other than minor works < 10 square feet. It should also be recognised that the cautious use of biocides and fungicides have almost no appreciable inclusion in any standards or guidelines.¹¹⁸ Most importantly the use of biocides has been seen to increase the cytotoxicity of the spores produced.¹¹⁹

7.7. Containment is a necessary action to prevent the spread of contamination.¹²⁰

7.8. Not all materials affected by mould growth can be salvaged and may require disposal¹²¹

7.9. Decontamination should be documented and sometimes assessed by third parties and confirmed with various forms of sampling where identified as suitable for quality control purposes.¹²²

7.10. It should be accepted that all listed decontamination protocols¹²³ recognise the importance of both visible and hidden mould. Most importantly some recognised agencies such as the AIHA state that all mould should be treated as a hazardous substance, and consequence, recommendations for decontamination, worker protection containment and disposal¹²⁴.

7.11. It should be recognised that in the referenced protocols for decontamination a separation between salvageable and non salvageable materials are discussed. Effective cleaning or decontamination is not always possible or cost effective¹²⁵

¹¹⁴ IOM Chapter 6 page 303

¹¹⁵ Harriet Burge and IOM Chapter 6 page 305

¹¹⁶ PPE Regulations 2009

¹¹⁷ IOM Chapter 6

¹¹⁸ IOM Chapter 6 pages 276 onwards.

¹¹⁹ IOM Chapter 2 page 71

¹²⁰ IOM Executive summary page 13

¹²¹ IOM Chapter 6 pages 273 -275

¹²² IOM Chapter 6 page 275

¹²³ IOM pages 226-227

¹²⁴ IOM Chapter 6 page 284

¹²⁵ IOM Chapter 6 Table 6-1

7.12. The basis of decontamination protocols listed¹²⁶ follows the identification of visible mould. From the content of the WHO¹²⁷ and IOM¹²⁸, it is clear that many other contaminants are likely to be hidden but present in water damaged buildings. These contaminants may be sub micron, non water soluble, non volatile, aerosolised almost continuously and may not effectively be removed by even meticulous cleaning.

7.13. Anecdotal evidence from the author of this review has shown that in seriously affected buildings where exposure has been chronic and occupants may have been sensitised that any amount of HEPA sandwich cleaning cannot remove the substances which cause allergenic, toxic or irritant effects¹²⁹.

7.14. The removal of dust or visible mould by vacuuming does nothing to remove aerosolised and inhalation of toxins or allergens presumed to be present and the effects are poorly understood.¹³⁰

7.15. It may not be possible to decontaminate all buildings or contents and moving out, re locating to a new property and disposing of contents may be the only way to relieve symptoms.¹³¹

8. Fallacies of Mould Health Risks

A publication from the esteemed John Hopkins Institute was cited as evidence about mould myths under the title of “**9 Common Mold Myths**”

8.1. Using the same stated sources of World Health Organisation and Institute of Medicine I have created reasonable doubt as to the accuracy of the articles assumptions or statements.

8.2. See full article

http://www.johnshopkinshealthalerts.com/reports/lung_disorders/2012-1.html

8.3. The John Hopkins article

Media reports have linked indoor mold exposure to everything from asthma to headaches. But what’s the real scientific evidence that exposure to mold in your home actually can cause physical symptoms? A recent review of scientific literature about mold-related diseases found that, while mold can cause certain health problems, many common claims just don’t hold up under scrutiny. Five allergists, including Robert A. Wood, M.D., of the Johns Hopkins University School of Medicine, set out to define what can and can’t be proved about mold exposure as laid out in the following nine issues.

¹²⁶ IOM Chapter 6 Table 6-1

¹²⁷ WHO Chapters 4-5

¹²⁸ IOM Chapter 2

¹²⁹ IOM Chapter 3 page 116

¹³⁰ IOM Chapter 3 page 93

¹³¹ IOM Chapter 5 page 232

8.4. Legal standing of the following John Hopkins article

This article has been discredited in both the medical fraternity and US courts due to various dubious references.

8.5. John Hopkins paper refers to a paper originating from the AAAAI¹³² Jay Portnoy who is a listed contributor is a doctor who did not know his name was on it.

8.6. An influential paper produced for the US Chamber “The Scientific Health Effects of Mold” was accredited to A. Saxon but he states he was not the author.

8.7. Another stated contributor Corren Robbins CIH was challenged by the judge as evidence on mycotoxin safety was extrapolated from animal studies which from a Kelly-Frye¹³³ (standard for admitting scientific evidence in a trial) hearing raised doubt as to accuracy and which subsequently became a foundation case law for removing all similar animal or rodent mycotoxin studies as a form of evidence in US courts.

8.8. The issue of white coat and collar experts denying the issues of mold health hazards has become a major legal issue as to why they are denying what the US Government has apparently accepted in the GAO¹³⁴.

8.9. The GAO published document of 2008¹³⁵ states “The American Academy of Pediatrics concluded in 2006 that a plausible link exists between acute pulmonary hemorrhage in infants and exposure to toxins that some molds produce”¹³⁶ More information on the legal standing of this document and indeed the court case which “apparently” discredits its authors is on line¹³⁷

8.10. Significant issues regarding the Kelly Order April 2006 Sacramento California

The following paraS (8.10.1) and (8.10.2) 8.10.3) are the appraisals of author (Jeff Charlton) regarding the lengthy documentation referred to in (8.10) The full case can be found by referencing Case number 02AS04291 Plaintiffs James Harold and D. Lee Harold, versus California Casualty Insurance Company and Westmont Construction Inc. Defendants.

8.10.1. Insurers legal team presented “scientific evidence ” that exposure of mycotoxins to rodents and animals proved exposure effects could be extrapolated to prove no health risk to humans existed.

¹³² American Academy of Allergy and Immunology

¹³³ <http://definitions.uslegal.com/f/frye-test/>

¹³⁴ Government Accountability Office

¹³⁵ <http://freepdfhosting.com/0dac523cb3.pdf>

¹³⁶ <http://freepdfhosting.com/0dac523cb3.pdf>

¹³⁷ <http://katysexposure.wordpress.com/2010/04/30/truth-out-sharon-kramer-letter-to-andrew-saxon-mold-issue/>

8.10.2. The Judge, Honorable Michael P. Kenny, of the Superior Court of California challenged the scientific evidence of rodent- human extrapolation on the grounds of Kelly-Frye¹³⁸ and in particular personal exposure levels, and the even greater and tenuous leap to modeling.

8.10.3. The Judge conclusion was:

The defence position that animal and rodent studies showing limited health effects from mycotoxin exposure could not be extrapolated to humans and that in future the courts would regard such evidence as being “a huge leap to go from a modeling theory to proof of lack of causation”

In a summation the Judge is reported as stating¹³⁹“My fundamental problem is in looking at it from a *Kelly-Frye* standpoint, I just didn’t see kind of acceptance in the scientific community with regard to what she (the expert) had done that would allow it to be sort of presented as such”

The Judge went on to state:

“Modeling has severe limitations, and one of the difficulties I was having here was this reliance upon animal studies to jump to a modeling conclusion generally with –again, I’m speaking from my own experience because there is nothing here in this transcript –generally one will use the data that one can receive either from animal exposure studies or other information to then input in a model to make a determination with some degree of reliability”, the judge continued, “Here I am not hearing any of those things. I am hearing essentially this jump from a literature review to a postulated model to a no harm result”.

8.10.4. Reference details and case excerpts can be found in Appendix Doc 1

TOXIC mold.

Popular reports about the health effects of mold are likely to include the term “toxic mold.” But that term can be misleading, the experts say. They point out that only certain mold spores produce toxins, and only under certain circumstances. Just because a particular mold can produce toxins doesn’t mean it will. Even if the mold is producing toxins, a person must breathe in a sufficient dose to be affected. It is highly ***unlikely*** that you could inhale enough mold in your home or office to receive a toxic dose

Response

¹³⁸ <http://www.genexdiagnostics.com/legal/witness>

¹³⁹ Mold Columns, Harris Martin Publishing, May 25, 2006

8.10.5. One nanogram mycotoxin is enough to cause adverse health effects in people. If we took the 10 nanogram threshold value for exposure to *Stachybotrys* spores at 3×10^{-15} at a respiratory rate of 30 m^3 per day it would require a background level of 100 spores m^3 to be inhaled for 1000 days.¹⁴⁰ There are other issues to consider, such as the presence of other spores in the air besides *Stachybotrys*, the possible presence of other toxins in the air, non-exposure related effects such as stress and psychological damage¹⁴¹, etc

8.10.6. References:

8.10.7. *Adverse Health Effects Associated with Molds in the Indoor Environment*, American College of Occupational and Environmental Medicine, October 27, 2002.

8.10.8. D.M. Khun and M.A. Ghannoum, *Indoor Mold, Toxigenic Fungi, and Stachybotrys chartarum: Infectious Disease Perspective*, Clinical Microbiology Reviews, January 2003, p144-172.

8.10.9. Adapted from Harriet A. Burge, *Health Effects of Biological Contaminants*, Indoor Air and Human Health, CRC Press, 1996, Chapter 10, p171-176.

8.10.10. 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,

8.10.11. 2006. <http://www.micotoxinas.com.br/boletim34.pdf> (PDF, 164kb) Air Resources Board: [How Much Air Do We Breathe?](#)

8.10.12. Study on mycotoxins suggests irritation could be a risk where spores are over $20,000 \text{ m}^3$. In this study¹⁴² restoration workers were identified as being at risk during decontamination works and both engineering controls and PPE are obvious controls required to protect both them and the occupants.

8.10.13. Study showing hotel workers who developed hypersensitivity pneumonitis from mycotoxin exposure¹⁴³

8.11. References:

¹⁴⁰ David Gallup CEO Aerotech laboratories co writer with Harriet Burge

¹⁴¹ Harriet Burge Aerotech publication

¹⁴² Harriet Burge Review of literature

¹⁴³ Trout et al 2001

- 8.11.1.** Trout D, Bernstein J, Martinez K, Biagini R, Wallingford K. 2001. Bioaerosol lung damage in a worker with repeated exposure to fungi in a water-damaged building. *Environmental Health Perspectives* 109:641-644
- 8.11.2.** Yike I, Distler AM, Ziady AG, Dearborn DG. 2006. Mycotoxin adducts on human serum albumin: biomarkers of exposure to *Stachybotrys chartarum*. *Environmental Health Perspectives* Aug 114(8):1221-6.
- 8.11.3.** Islam Z, Harkema JR, Pestka JJ. 2006. Satratoxin G from the black mold *Stachybotrys chartarum* evokes olfactory sensory neuron loss and inflammation in the murine nose and brain. *Environmental Health Perspectives* 114(7):1099-107
- 8.11.4.** Rosenblum Lichtenstein JH, Molina RM, Donaghey TC, Brain JD. 2006. Strain differences influence murine pulmonary responses to *Stachybotrys chartarum*. *Am J Respir Cell Mol Biol.* (Epub ahead of print)
- 8.11.5.** Kelman BJ, Robbins CA, Swenson LJ, Hardin BD. 2004 Risk from inhaled mycotoxins in indoor office and residential environments. *International Journal of Toxicology* 23:3-10.

8.12. In the UK it is not at all unusual to find *Stachybotrys* in water damaged buildings without visible mould but with spore counts in the low thousands per cubic meter and the occupants are often exposed for months to years. Under the rational of UK and EU health and safety legislation coupled to the Housing Health and Safety Rating Regulations it can be seen that a significant health risks exists from what is described in the HHSRS¹⁴⁴ 2004 as a significant hazard.

8.13. All moulds under proper conditions are capable of eliciting a negative health response in humans, such as inflammations allergy or infection¹⁴⁵.

8.14. The HHSRS requires a threat assessment¹⁴⁶ of environmental conditions and specifically mould hazards covering current and conditions possible changes in the threat or potential of mould for up to 12 months¹⁴⁷ following inspection. This assessments includes risks of harm associated with physical and mental health (stress)¹⁴⁸

8.15. For the purposes of harm or hazard the risk and hazard assessments of the HHSRS may include assessments of hazard and harm which may be substantiated by medical records or hospital admission¹⁴⁹

¹⁴⁴ Housing Health and safety Rating System 2004. An enforceable regulation protecting tenants

¹⁴⁵ RICS Royal Institute of Chartered Surveyors Risk of mould damage over the whole life of a building 2003
http://www.rics.org/site/download_feed.aspx?fileID=2915&fileExtension=PDF

¹⁴⁶ HHSRS Chapter 1 1.06

¹⁴⁷ HHSRS Chapter 1 2.20

¹⁴⁸ HHSRS Chapter 1 2.09

¹⁴⁹ HHSRS Chapter 1 2.11

- 8.16.** The article states “unlikely” risk in terms of health and safety but this is not the same as never and in terms of risk and liability the question of what is likely or unlikely can be assimilated to the legal interpretation of reasonable. With a national population of atopic public ranging from 5% to 40% we have a risk factor of susceptible people in the UK of between 3 million and 24 million depending on individual and unknown susceptibility and dose or levels/period of exposure.
- 8.17.** The toxic dose level has not been established and can vary in individuals¹⁵⁰ and effects of exposure can be from acute or chronic exposure, making measurement of exposure response immeasurable.
- 8.18.** Toxicity caused by synergistic effects of differing and accumulated toxins present is unknown¹⁵¹
- 8.19.** The water damaged (damp) building can be expected to have a combination of chemical biological and allergenic components¹⁵², of which the toxic effects or levels of maximum levels of exposure have yet to be assessed or proven, the presence of these toxins may result in a synergy greater than the individual sum total which is unknown,(Risk)
- 8.20.** Except for a few studies on cancer, mycotoxin studies have revolved around short term exposure of high concentrations, which may not reflect the risk of chronic exposure to humans even at low doses¹⁵³
- 8.21.** Little information is available regarding the degree of mycotoxin exposure by building occupants¹⁵⁴
- 8.22.** The level of dose required to adverse health effects in humans has not been determined¹⁵⁵ That doesn't mean its low risk it means the research work hasn't been completed however large scale studies from Germany, Canada, Minnesota, have shown that when spore concentrations exceed 75, 100,000 cfu/g of dust there is an increased incidence of respiratory health problems. The Czech republic recognises that >2000,cfu/m³ is related to increased health problems.
- 8.23.** These are levels at which increased acute health effects are measurable. What this means is that sub acute health effect are occurring at lower levels. Hence, Finland, Germany, Singapore, Brazil, Czech, Portugal, WHO, Canda, ACGIH (proposed), NIOSH (proposed) and others all applied a safety factor and recommend exposure levels not exceed 200-500 cfu/m³.¹⁵⁶

¹⁵⁰ IOM Chapter 4 P170

¹⁵¹ IOM Chapter 3 p94

¹⁵² IOM Chapter 3

¹⁵³ IOM Chapter 4 p125

¹⁵⁴ Sorenson et al 1987 IOM Chapter 4 p71

¹⁵⁵ IOM Chapter 4 p170

¹⁵⁶ Robert Brandy CIH PhD World Wide Exposure Standards for mould and Bacteria

8.24. The identity of people vulnerable (atopic) to exposure is not known ¹⁵⁷

8.25. It is accepted that some mould can produce toxins under certain circumstances¹⁵⁸, and these are often present in water damaged homes¹⁵⁹, These circumstances are acknowledged as during growth and challenges from other moulds and bacteria¹⁶⁰ or when faced with attack from desiccation or the application of fungicides¹⁶¹ and generally during sporulation¹⁶².

8.26. The moulds recognised as producing these moulds are scientifically referred to as toxigenic and typically include the moulds found to grow in water damaged buildings and include several species of Aspergillus, Penicillium, Fusarium, and Stachybotrys¹⁶³

8.27. The toxins and symptoms of exposure known to be associated with the above moulds include the following neurotoxins which are classified as:

8.27.1. Tremorogenic,

8.27.2. Paralytic,

8.27.3. Neurotoxic transmitters that target organs¹⁶⁴

8.28. Symptoms of exposure are explained as, partial paralysis, tremors, convulsions, limb weakness, ataxia (uncoordinated movement)gastrointestinal effects, acute cardiac beriberi, brain and retinal issues, suppression of immune system, degeneration of the central nervous system, cerebral toxicity¹⁶⁵

8.29. The term “unlikely to inhale sufficient toxins to cause a toxic dose” cannot be substantiated or identified because:

8.29.1. Persons both atopic and non atopic exposed to mould and bacteria may display symptoms not generally associated with allergic response and this includes, nervous system effects, suppression of immune response, hemorrhage of mucous membrane of the intestinal and respiratory tract ,rheumatoid disease, and loss of appetite^{166,167}.

8.29.2. The genetic predisposition of those exposed is unknown. Section to be added later and not part of the PCA review.

¹⁵⁷ IOM Chapter 6 p287

¹⁵⁸ IOM Chapter 1 p66-68

¹⁵⁹ IOM Damp Indoor Spaces and Health Chapter 4

¹⁶⁰ IOM Chapter 1 p70

¹⁶¹ IOM Chapter 1 p71 Murtoniemi et al 2003

¹⁶² IOM Chapter 4 P165

¹⁶³ Table 4-1 Chapter 4 IOD

¹⁶⁴ IOM Table 4-4 P160

¹⁶⁵ IOM Chapter 4 P160-168

¹⁶⁶ IOM Chapter 4 p125

¹⁶⁷ WHO Executive summary

- 8.29.3.** There is a variable and uncertain relationship between spore levels and allergens, toxins, and irritant content ¹⁶⁸
- 8.29.4.** Some moulds “Aspergillus” species known to grow in water damaged buildings may (risk) produce mycotoxins associated with cancer but no studies on potential carcinogenic role have been done ¹⁶⁹
- 8.29.5.** The size of the affected area is unknown and spore size variation and aerosolisation “drop out” will influence occupants exposure ¹⁷⁰ again influenced by secondary aerosolisation ¹⁷¹ or re suspension of culturable and non viable spore fragments ¹⁷²
- 8.29.6.** Height or age of occupants will affect exposure ¹⁷³ In summary the whole process of inhalation exposure is poorly understood ¹⁷⁴
- 8.29.7.** Some mould mycotoxins slow the ciliary beating of the respiratory system causing spores and fragments to be inhaled deeper than otherwise expected ¹⁷⁵ and also affect the alveolar macrophages response which usually destroy toxic or allergenic particles. ¹⁷⁶
- 8.29.8.** Research has shown that moulds that can produce mycotoxins under appropriate environmental and competitive conditions ¹⁷⁷, can and do grow indoors and damp indoor spaces which may also facilitate the growth of bacteria that can have a toxic, and inflammatory effect. Furniture and building materials are also considered to have the potential to release toxins but little information is available ¹⁷⁸.
- 9. Mold and Asthma.** While allergic responses to inhaling mold are a recognized factor in lower airway disease such as asthma, studies show that outdoor mold is more likely to cause problems for asthmatics than mold found indoors. A better assessment of the effects of indoor mold on people with asthma would require studies that follow people over a long period and take into account factors that could affect the results, such as humidity and other airborne allergens and irritants.

Response

¹⁶⁸ IOM Chapter 3 p115-116

¹⁶⁹ IOM Chapter 4 p169n

¹⁷⁰ IOM Chapter 3 Exposure p 92

¹⁷¹ IOM Chapter 3 p93

¹⁷² IOM Chapter 3 p93

¹⁷³ IOM chapter 3 p93

¹⁷⁴ IOM chapter 3 p93

¹⁷⁵ IOM Chapter 4 page 150

¹⁷⁶ IOM Chapter 4 page 150

¹⁷⁷ IOM Chapter 4

¹⁷⁸ IOM Executive summary page 7

- 9.1.1.** Sub micron particles of mould spores contaminated with allergens are released which can enter the deep lung and alveoli¹⁷⁹ (where blood oxygen exchange occurs) this may have disproportionate dose/effect to normal exposure data although it has not been studied.
- 9.1.2.** Once deposited in the lungs the agents may react with biomolecules and be absorbed into the blood, the nature of interaction between inhaled agents and the human body remain uncertain and this limits the ability to define dose¹⁸⁰.
- 9.1.3.** The potential for asthma sufferers to have worse symptoms when outdoors has no bearing to those with no previous asthma history but who do develop asthma from mould exposure due to possible sensitivities.
- 9.1.4.** The exacerbation of asthma from mould exposure may take between 20 minutes to 48 hours.¹⁸¹
- 9.1.5.** Water damaged buildings result in the amplification of many biological agents including bacteria and mould and in these damp circumstances there is a strong consistent indicator of risk of asthma, respiratory symptoms WHO¹⁸²
- 9.1.6.** International studies provide sufficient epidemiological evidence to show occupants of mould affected buildings are at increased risk of respiratory symptoms, respiratory infections, exacerbation of asthma. WHO¹⁸³
- 9.1.7.** There is clinical evidence that exposure to mould increases the risk of hypersensitivity pneumonitis, which is supported by toxicology evidence from in vivo and in vitro studies. WHO¹⁸⁴
- 9.1.8.** The international and increasing prevalence of asthma and allergies increase the atopic population¹⁸⁵
- 9.1.9.** There is a strong epidemiological intervention study with other studies which suggests dampness and mould exacerbates asthma in children. There is also sufficient epidemiological evidence of association between damp buildings or mould and asthma development, asthma exacerbation and upper respiratory infections except otitis media an upper respiratory infections such as cough wheeze and dyspnoea.¹⁸⁶

¹⁷⁹ IOM Chapter 2 page 66

¹⁸⁰ IOM Chapter 3 page 94

¹⁸¹ IOM Chapter 3 page 91

¹⁸² WHO Executive summary

¹⁸³ WHO Executive summary

¹⁸⁴ WHO Executive summary

¹⁸⁵ WHO Executive summary

¹⁸⁶ WHO 4.4

9.1.10. Mould and damp buildings may be responsible for 21% of asthma in the USA¹⁸⁷

9.1.11. Molds can trigger asthma attacks in persons who are allergic (sensitized) to molds. The irritants produced by molds may also worsen asthma in non-allergic (non-sensitized) people¹⁸⁸

9.1.12. Molds can trigger asthma attacks in persons who are allergic (sensitized) to molds. The irritants produced by molds may also worsen asthma in non-allergic (non-sensitized) people

9.1.13. Similarly, we still don't know exactly how or why cigarette smoking causes cancer. It is only a **relationship**. See why a smoker lost his case against Imperial tobacco, He might have got cancer if he didn't smoke.

<http://www.internationallawoffice.com/Newsletters/Detail.aspx?r=11694&redir=1>

10. Mold and Allergies. The link between mold and allergies is even weaker, the experts say. Current research doesn't provide a persuasive case that exposure to mold in the outdoor air plays a role in allergies, and studies linking indoor molds to upper airway allergy are even less compelling.

Response

10.1.1. Where conditions for mould growth occur, microbial growth may result in the amplification of spores cell fragments, allergens mycotoxins, endotoxins,β glucans and Volatile Organic Compounds in indoor air. While the causative agents of adverse health effects have not been identified conclusively, an excess of any of these agents is a potential health hazard. WHO¹⁸⁹

10.1.2. Exposure to microbial contamination and resultant health issues cannot be quantified precisely. No quantitative health based guideline values or thresholds can be recommended for micro organisms.WHO¹⁹⁰

10.1.3. Low income families in poor housing are seen as a priority in remediation of mould in order to reduce the additional health burden, clearly accepting the additional risk.¹⁹¹

10.1.4. Exposure to microbial contamination associated with damp buildings is clinically associated with respiratory symptoms, allergies, asthma and immunological reactions. WHO¹⁹²

¹⁸⁷ WHO section 4.4

¹⁸⁸ Environmental Protection Agency http://www.epa.gov/mold/append_b.html#Irritant_Effects

¹⁸⁹ WHO Executive summary

¹⁹⁰ WHO Executive summary

¹⁹¹ WHO Executive summary

¹⁹² WHO section 1.1

10.2. Mold and Skin Rashes. Exposure to molds doesn't contribute to atopic dermatitis, or rashes.

Response

10.2.1. The US Centre for Disease Prevention and Control state that some people are sensitive to moulds and can be affected by various irritations including skin.¹⁹³

10.2.2. Mold exposure can cause irritation of the eyes, skin, nose, throat, and lungs, and sometimes can create a burning sensation in these areas¹⁹⁴

11. Mold and Sinusitis. There's no clear-cut evidence that sensitivity to mold causes chronic sinusitis, nor are there conclusive data to show that mold-killing antifungal drugs such as amphotericin, applied to the nasal passages, are an effective treatment for sinusitis.

11.1.1. There have been no specific studies which can be used to disprove or confirm association, however fungi are commonly present from the nose secretions of those suffering from sinusitis¹⁹⁵.

11.1.2. It should be noted that "inadequate or insufficient evidence to determine whether an association exists" does not rule out the possibility of an association. Rather, it means that no studies examined the relationship or that published study results were of insufficient quality, consistency, or statistical power to permit a conclusion about an association¹⁹⁶

12. Mold and Infection.

Superficial fungal infections, such as toenail fungus or jock itch, generally result from fungi that develop inside the warm, moist environments found in shoes or tight garments. Thrush can develop inside the mouths of people with weakened immune systems, such as those who have AIDS or cancer. These infections generally are not the result of exposure to mold in the home or workplace

Response

12.1.1. Tricothecenes from *Stachybotrys* affected workmen handling contaminated materials resulting in painful skin lesions hands armpits and genitals.¹⁹⁷

12.1.2. The level or strength of the immune system of those exposed is unknown and this significant factor can be heightened by the exposure to some mycotoxins which effectively turn off or reduce the immune system¹⁹⁸

¹⁹³ http://www.cdc.gov/mold/dampness_facts.htm

¹⁹⁴ Environmental Protection Agency http://www.epa.gov/mold/append_b.html#Irritant_Effects

¹⁹⁵ IOM chapter 5 pages 208-211

¹⁹⁶ Mold Prevention strategies and possible health effects, CDC Mold Work Group supported by OSHA and US Protection Agency

¹⁹⁷ IOM Chapter 4 page 166

12.1.3. When moisture problems occur and mold growth results, building occupants may begin to report odors and a variety of health problems, such as headaches, breathing difficulties, skin irritation, allergic reactions, and aggravation of asthma symptoms; all of these symptoms could potentially be associated with mold exposure.

13. Mold and Irritation.

Mold found indoors, even inside damp buildings, is not likely to cause irritation of the eyes or throat -- and if it does, the effects are short-lived. Symptoms or signs persisting weeks after exposure and those accompanied by complaints related to the nervous system, brain, or whole body (such as those attributed to chronic fatigue) can't be pinned on the irritant effects of mould exposure.

Response

13.1. Inhaling or touching mold or mold spores may cause allergic reactions in sensitive individuals. Allergic reactions to mould are common - these reactions can be immediate or delayed. Allergic responses include hay fever-type symptoms, such as sneezing, runny nose, red eyes, and skin rash (dermatitis). Mold spores and fragments can produce allergic reactions in sensitive individuals regardless of whether the mold is dead or alive. Repeated or single exposure to mold or mold spores may cause previously non-sensitive individuals to become sensitive. Repeated exposure has the potential to increase sensitivity¹⁹⁹

13.1.1. Bacteria and mould can produce primary metabolites such as MVOCS and VOCS which can cause irritation to eyes and skin²⁰⁰

14. Mold and Immune System Damage.

There is no credible evidence to suggest that environmental exposure to mold damages the immune system. The experts warn against immune-based tests given to look for intolerance to mold and other substances in the environment—so-called multiple chemical sensitivity. The authors specifically advise against using blood tests that look for a wide range of non-specific changes in the immune system. They also discourage using tests of auto antibodies, which are abnormal antibodies that the body sometimes produces in reaction against its own tissues. These tests are expensive and do not provide useful information that will help to diagnose or manage diseases related to mold, they say.

Response

14.1.1. Symptoms of exposure are explained as, partial paralysis, tremors, convulsions, limb weakness, ataxia (uncoordinated movement)gastrointestinal

¹⁹⁸ IOM Chapter 4 p163 Gliotoxin

¹⁹⁹ Centre of Disease Control Mold Work group reviewed by USA Environmental Protection Agency and Occupational Health Administration

²⁰⁰ IOM Chapter 4 page 165

effects, acute cardiac beriberi, brain and retinal issues, suppression of immune system, degeneration of the central nervous system, cerebral toxicity²⁰¹

14.1.2. Some investigators have suggested respiratory infections including sinusitis, common cold, otitis and bronchitis may be due to immunosuppressive effect.

²⁰²

14.1.3. Symptoms associated or similar to Chronic Fatigue Syndrome are associated with mycotoxin exposure.²⁰³

15. Mold and Hypersensitivity Pneumonitis. This uncommon inflammation of the lungs, an example of which is Farmer's Lung, is caused by exposure to an allergen, usually organic dust that may come from animal dander, molds, or plants. A person generally develops this condition only after high-dose or prolonged exposure, or both, to mold or other allergens.

Much of the hoopla over mold exposure came in the wake of Hurricane Katrina, the experts note in their report, which appeared in the *Journal of Allergy and Clinical Immunology*. The flood-ravaged areas of the Gulf Coast, sadly, have provided a natural laboratory, which enables medical researchers to address lingering questions about the health effects of mold.

Response

15.1.1. The confirmation or diagnosis of HP is difficult as there are no single characteristic findings. The test for circulating immunologic antibodies has been shown to have little value as a marker for chronic HP but it is generally agreed they are better used as indicators of recent high exposure to moulds and thermophilic actinomycete antigens, (fungi like bacteria).

15.1.2. Fungi such as *Trichosporon* is a recognised form of seasonal mould causing HP. Mould decontamination of buildings is a normal requirement alongside standard medical therapy. In some cases mould decontamination isn't successful and the only recourse is for those affected to move away from the property.²⁰⁴

15.1.3. Where uncertainties regarding scientific knowledge exist, practical applications should be used to protect the public health²⁰⁵ Sufficient evidence exists of association between several adverse health outcomes and exposure to materials contaminated by mould.

16. John Hopkins Review Ends

²⁰¹ IOM Chapter 4 P160-168

²⁰² IOM Chapter 5 page 237

²⁰³ IOM Chapter 5 page 246

²⁰⁴ IOM Chapter 5 page 232

²⁰⁵ Centre of Disease Control Mold Work group reviewed by USA Environmental Protection Agency and Occupational Health Administration

17. Insurance and insurers/contractors responsibilities

It can be seen from the foregoing that mould is a potential health hazard and cause of material, latent, or long term damage. From the authors work as an expert witness and extensive inspection and reviews of contractors remediation works, there are serious concerns regarding the lack of attention to drying and or decontamination of water damaged or damp buildings.

Typically investigation follows newly reported and recorded health effects of building issues such as visible mould, odour and invariably point to building design or construction defect or contractor incompetence in restoration or remediation.

Considering the extent of information and evidence available it may surprise some that currently in the UK, there are no recognised standards or guidelines of drying, decontamination and indeed no nationally recognised training or certification in either subject.

Unfortunately I have yet to see a meaningful drying or sanitation /completion certificate issued for any flood affected or water damaged building. Responsibility and potential liability of the builder and or insurer regarding personal injury and material damage may therefore extend over many years following a water damage event.

17.1.1. Insurance issues and liability²⁰⁶

The insurance perspective is three-fold:

17.1.1.1. Damage to users/occupiers' health

17.1.1.2. Damage to the health of the site workers, resulting from mould growth in existing and new properties

17.1.1.3. Damage to property from mould growth

17.1.2. The Institute for International Research has predicted that insurance premiums in the US are to rise by 40% to offset claims for mould damage

17.1.3. *“The insurance industry is looking at mold as being the next asbestos They are looking to get an absolute exclusion.”*

17.1.4. Insurance companies are more worried about the health aspects of mould rather than property damage; this means they may exclude physical injury and offer some property damage coverage; albeit to maximum low level of liability.

²⁰⁶ University of Reading, RICS Foundation The risk of mould damage over the whole life of a building 2003

18. Is the building safe?

Following a water damage event²⁰⁷ it might reasonably be expected that a competent person, engaged to assess damage or restoration would provide any one of the following “substantiated” statements:

- 18.1.1. This water damaged building is not a health hazard
- 18.1.2. This water damaged building is unlikely to be a health hazard
- 18.1.3. This water damage building might be a health hazard to some people (Who we can’t usually identify until they get sick)
- 18.1.4. This water damaged building is a potential health hazard
- 18.1.5. This water damaged building is a health hazard
- 18.1.6. I don’t know if the building is safe I will seek competent advice

18.2. The health hazards and risks of water damaged buildings have been recognised for many years and these questions can reasonably be placed retrospectively to duty holders or those with responsibilities to ensure compliance with legislation and civil duty of care.

19. Conclusion

- 19.1.1. A water damaged building may not be an initial health hazard but the consequence of bio amplification over time substantially increases the potential hazard. It is therefore imperative that steps are taken to show that adequate steps were taken to minimise risk and hazard?
- 19.1.2. The forgoing shows the complex issues of individual organisms producing toxins and allergens but frequency is unknown. The synergistic effects of these separate substances may increase the sum total hazard which is often unknown until symptoms appear. It can be seen as a duty of care exists to assess these risks and record findings.
- 19.1.3. The reality is that these toxins and allergens are difficult to measure although some standards do exist²⁰⁸ and it is impossible to assess individual exposure and differing personal sensitivity. As a recognised hazard and potential risk exists in a WDB it may be seen as a requirement to show how these issues were assessed and what steps were taken to reduce possible exposure.
- 19.1.4. There are no currently available levels of permitted exposure even to complex chemical compounds. The toxic soup found in water damaged buildings may be impossible to assess. This of course does not mean a hazard is not identifiable, but it does mean it may be difficult to quantify in terms of personal risk. Absence of evidence is not evidence of absence and while science may not be available to quantify risks, legislation can. It may be seen as a duty of care and legal requirement to show how assessments were or are made in WDBs.

²⁰⁷ Water damage includes damp and condensation IOM Executive summary page 4

²⁰⁸ Endotoxin Units

19.1.5. Where a possible hazard exists, all reasonable care should be used to limit that potential risk. Where a known but unquantifiable hazard exists it should be assumed to be a high risk unless controlled, reduced or eliminated. Contractors and their insurers or those that nominate contractors should be fully aware of these implications and liabilities.

19.1.6. Contractors , insurers and landlords may be responsible or liable for the health effects and material damage to properties and occupants previously involved with or exposed to water damage buildings. This is especially relevant in poorly remediated or maintained buildings where duty holders have failed to address or identify the potential hazards, and or provide relevant documentation.

Review Ends

Please make comments to email below and state if you wish to be included on the review panel.

Contributions or supportive comment to this article received from :

- **Harriet Burge PhD**
- **Robert Brandy PhD CIH**
- **Sharon Noonan Kramer**

Jeff Charlton

www.buildingforensics.co.uk

email forensic@999team.org

Phone 07990 500 999

Appendix

Doc 1 Provided by Sharon Noonan Kramer

Are you aware of the Kelly Order, April 14, 2006, Sacramento, CA? It is an issue changing significant finding that will remove 'road blocks' and allow the medical understanding of mold induced illnesses to more easily go forward.

The Kelly Ruling is a huge blow to those who are most concerned about perpetuating the litigation defense myth of serious mold illnesses do not occur from exposure within an indoor environment. The Ruling discredits the entire foundation of All the medical associations, government documents, etc, that illness from inhaling mycotoxins indoors is "not plausible, improbable and junk science". One could say those, who are more concerned of financial liability than they are of the lives and safety of others, just got a "dose" of their own medicine at a "level of which we see effects".

The significance of this Kelly Ruling as it pertains to mold litigation is:

The defense argument of "not plausible, improbable and junk science" has now been determined by the courts to be "not plausible, improbable and junk science".

Case # 02AS04291, James Harold and D. Lee Harold, Plaintiffs vs. California Casualty Insurance Company and Westmont Construction, Inc., Defendants

Honorable Michael P. Kenny, Judge of the Superior Court of California, County of Sacramento

The Plaintiffs were represented by Peter Alfert, Attorney at Law; Michael J. Cochrane, Attorney at Law, and Karen Kahn, Attorney at Law.

The Defendant, California Casualty Insurance Company, was represented by Stephen M. Hayes, Attorney at Law, and Robert S. McLay, Attorney at Law.

The Defendant, Westmont Construction Company, was represented by Ronald E. Enabnit, Attorney at Law.

Jury award to plaintiffs: \$2.3 Million.

Subject paper deemed not acceptable by Kelly Ruling in the case, April 14, 2006

Title: Risk from inhaled mycotoxins in indoor office and residential environments. Int J

Toxicol 2004; 23: 3-10.

Robbins CA, Swenson LJ, Hardin BD (Principals of litigation defense support corp.

Veritox, Inc and formerly named GlobalTox, Inc.)

Slang: Veritox, 2004

The above is the review piece that was found not to be based upon sound science and therefore not to be presented in the court before a jury. The judge found it to be a "huge leap", for PhD's to take rodent studies, apply a little math and then write a review that all human illness is not plausible from mycotoxin inhalation within an indoor environment. Dr. Robbins of Veritox, Inc., could not cite anyone else's research or review paper that made the same conclusion.

The reason for this is because there are not any.

Mold Columns

Harris Martin Publishing

May 25, 2006

....Defendants called Andrew Saxon, M.D., of UCLA Medical School; and Coreen A. Robbins, MHS, Ph.D., CIH of Veritox in Redmond, Wash.

Robbins countered plaintiffs' experts' opinions on mold hazards and the remediation procedures and opined that the couple could have moved back into the house after Westmont's repair work was completed.

Judge Kenney held a *Kelly-Frye* hearing before trial and limited Robbins's testimony by precluding any reference to animal studies of mold hazards.

Reviewing Robbins' deposition testimony, Judge Kenney concluded that the basis for her testimony on mycotoxins and human exposure was a literature review, which he found insufficient.

'Also, when I reviewed the DHS report from April of 2005, DHS, Department of Health Services was talking about the fact that they were unable to establish personal exposure levels at this point in time based on a lack of sufficient information, and yet Dr. Robbins is asking to take an even greater step and go beyond establishing, for example, a personal exposure level and jump to modeling, which is far more tenuous and far more unreliable even in establishing something that is as hard as a personal exposure level. So those are the difficulties I'm having with Dr. Robbins' testimony,' Judge Kenney said.

The judge said that he is familiar with the use of animal studies and derivative models for humans and that such models are commonly accepted in the scientific community, but he said he is not sure such models for mycotoxin exposure would pass a *Kelly-Frye* test for admissibility.

'My fundamental problem is in looking at it from a *Kelly Frye* standpoint I just didn't see kind of acceptance in the scientific community with regard to what she had done that would allow it to be sort of presented as such,' Judge Kenney said.

'Modeling has severe limitations, and one of the difficulties I was having here was this reliance upon animal studies to jump to a modeling conclusion generally with — again, I'm speaking from my own experience because there is nothing here in this transcript — generally one will use the data that one can receive either from animal exposure studies or other information to then input in a model to make a determination with some degree of reliability,' the judge continued. 'Here I'm not hearing any of those things. I'm hearing essentially this jump from a literature review to a postulated model to a no harm result'

To understand why this is such a boon to move the medical science forward and why it is such a significant ruling - that dispels the myth of serious mold induced illnesses are not occurring, one has to go back to the year 2000:

2000

Title: Health effects of mycotoxins in indoor air: a critical review. Appl Occup Environ Hyg.2000;15:773-84.

Robbins CA, Swenson, L.J., Nealley, M.L., Kelman, B.J. and Gots, R.E.

Slang: Veritox, 2000

Robbins, Swenson and Kelman - Principals in defense litigation support corp, Veritox.

Nealley and Gots -Defense experts with International Center for Toxicology and Medicine.

Veritox 2000 is based on the same premise as the Veritox 2004 cited above. Rodents, authors added math, human illness not plausible.

2002

The American College of Occupational and Environmental Medicine (ACOEM) Mold Statement

Title: Adverse Human Health Effects Associated with Molds in the Indoor Environment
October 27, 2002

Kelman BJ (Veritox), Hardin BD (Veritox), Saxon AJ.(University of California - UC)
Edited & published in the Journal of ACOEM, the JOEM 2003

Slang: ACOEM MS, 2002

"Levels of exposure in the indoor environment, dose-response data in animals, and dose-rate considerations suggest that delivery by the inhalation route of a toxic dose of mycotoxins in the indoor environment is highly unlikely at best, even for the hypothetically most vulnerable subpopulations."

Sole reference for the above statement:

Veritox, 2000. Reference 63

NONE of the other 83 references cited for this 'state of the art review piece' support the above conclusion.

ACOEM MS, 2002 was presented as a position statement purportedly representative of 7000 physicians' understanding of mold/mold toxin induced illness. ACOEM is made up primarily of physicians who evaluate injured workers on behalf of insurers and employers.

3

US Chamber of Commerce/Center for Legal Policy -Manhattan Institute Mold Statement

“Center for Legal Policy is a leading voice for reform of America’s civil justice system.” according to their website.

Title: A Scientific View of the Health Effects of Mold

Bryan Hardin, PhD (Veritox), Andrew Saxon MD (UC), Correen Robbins, PhD, CIH

(Veritox) and Bruce J. Kelman, Ph.D., DABT (Veritox)

Slang: USCC MS, 2003

“Thus the notion that ‘toxic mold’ is an insidious secret ‘killer’ as so many media reports and trial lawyers would claim is ‘Junk Science’ unsupported by actual scientific study.”

Sole references for the above statement:

Veritox, 2000 and ACOEM MS 2002

The USCC MS 2003 has been reported by the Veritox authors to be a "lay translation" of the ACOEM Mold Statement. They were ‘commissioned’ by the political think-tank, the Manhattan Institute to write this lay translation. The authors received \$40,000 for interpreting the national protocol writing, medical association’s (ACOEM) understanding to mean that all mold illness is based upon ‘Junk Science’. It was then shared with stakeholder industries (real estate, building, mortgage and insurance) in a fanfare presentation in Washington, DC, July 17, 2003.

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2003

National Association of Realtors (NAR)

Title: Moldy Claims: The Junk Science of Toxic Mold

Kelman BJ.(Veritox) Hardin BD.(Veritox) Saxon AJ.(UC)

Slang: NAR 2003

*“Thus the notion that ‘toxic mold’ is an insidious secret ‘killer’ as so many media reports and trial lawyers would claim is ‘Junk Science’ unsupported by actual scientific study.”*

Sole references for the above statement:

Veritox, 2000, ACOEM MS 2002 and USCC MS 2003.

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2004

Title: Risk from inhaled mycotoxins in indoor office and residential environments. Int J

Toxicol 2004; 23: 3-10.

Robbins CA, Swenson LJ, Hardin BD. (Veritox, Inc. Principals)

Slang: Veritox, 2004

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2003 to 2005

Various Government Regulatory (CDC & EPA), Medical Associations (ACAAI, SOT), Industrial Hygiene Associations (AIHA), etc. make the findings of "not plausible" citing Veritox 2000, ACOEM MS 2002, USCC MS 2003, NAR 2003 and/or Veritox 2004. These five review papers have been cited as authoritative documents by the defense in virtually every mold litigation case in the US.

~~~~~  
2005

Example of Impact on the Courts

Testimony of Bruce J. Kelman, President of Veritox, Inc.

Author of Veritox 2000, ACOEM MS 2002, USCC MS 2003, NAR 2003 & Co-principal Veritox 2004

February 18, 2005, Haynes vs. Adair Homes, Inc. Case No. CCO211573,

In the Court of the State of Oregon.

"Based on the studies that you have done, the literature that you have discussed, and your experience and training, have you formed an opinion based on reasonable scientific probability or certainty as to whether or not there was enough mycotoxin in the home to have caused any illness to Mrs. Haynes, Michael Haynes, or Liam Haynes?" Dr. Kelman's answer: "Yes." The attorney: "And, what is that opinion, doctor?" Kelman: "There could not be. I mean, the differences between the maximum dose that we could come up with and the level at which we see effects for a broad range of mycotoxins is just too great."

~~~~~  
2006

American Academy of Allergy, Asthma and Immunology (AAAAI) Mold Position

Title: The medical effects of mold exposure

Bush RK, Terr A.(UC), Saxon AJ (UC) and Wood RA.

Slang: Quad AI 2006

"Calculations for both acute and subacute exposures on the basis of the maximum amount of mycotoxins found per mold spore for various mycotoxins and the levels at which adverse health effects are observed make it highly improbable that home or office mycotoxin exposures would lead to a toxic adverse health effects.<sup>1</sup>, 29

Thus we agree with the American College of Occupational and Environmental Medicine evidence-based statement and the Institute of Medicine draft, which conclude that the evidence does not support the contention that mycotoxin-mediated disease (mycotoxicosis) occurs through inhalation in nonoccupational settings."

Sole reference for the above statements:

ACOEM MS 2002 - Reference 1; Veritox 2004 - Reference 29.

Note: Saxon (UC) is an author of ACOEM MS 2002, USCC 2003, NAR 2003, & Quad AI 2006

Veritox principals are authors of Veritox 2000, ACOEM MS 2002, USCC 2003, NAR 2003 & Veritox 2004.

~~~~~  
2006

Robbins Order, Kelly Ruling, April 14, 2006

Veritox 2004 does not pass Kelly.

Veritox 2004 is the 'second generation' of Veritox 2000. Both 'review papers' are founded on the same premise that is now debunked as not being of sound scientific protocol to determine absence of human illness from mycotoxin inhalation indoors.

ACOEM MS 2002, USCC MS 2003, NAR MS 2003, and Quad AI MS 2006 are all founded on the Veritox 2004 or Veritox 2000.

Statements of "*not plausible, improbable, and junk science*" within all papers are debunked by the debunking of the Veritox 2004.

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Additional Information of Significance, 2006

The Institute of Medicine (IOM), Damp Indoor Spaces and Health Report, was a primary exhibit in the Kelly hearing that discredited the Veritox 2004.

IOM Executive Summary:

"Toxicologic studies, which examine such responses using animal and cellular models, cannot be used by themselves to draw conclusions about human health effects."

IOM Chapter 4 Mycotoxins

Summary:

"Except for a few studies on cancer, toxicologic studies of mycotoxins are acute or short-term studies that use high exposure concentrations to reveal immediate effects in small populations of animals. Chronic studies that use lower exposure concentrations and approximate human exposure more closely have not been done except for a small number of cancer studies."

IOM Chapter 4 Mycotoxins

Summary

Considerations in Evaluation of Evidence

"Most of the information reviewed in this chapter is derived from studies in vitro (that is studies in an artificial environment, such as a test tube or a culture medium) or animal studies. In vitro studies, as explained below, are not suitable for human risk assessment. Risk can be extrapolated from animal studies to human health effects only if chronic animal exposures have produced sufficient information to establish no-observed-adverse-effect levels (NOAELs) and lowest-observed-adverse-effect levels (LOAELs). Extrapolation of risk exposure from animal experiments must always take into account species differences between animals and humans, sensitivities of vulnerable human populations, and gaps in animal data."

2006

Minutes from the US Surgeon General's Workshop on Indoor Air are published

"Dr. Noreen Clark [Chair of the IOM Damp Indoor Spaces and Health Report, 2004] indicated that the report did not consider only respiratory symptoms, but that these were the symptoms for which associations were strongest. *She noted that "absence of evidence is not evidence of absence," and said that the report did not intend to dismiss the possibility of effects for which the existing evidence of association was not strong or for which evidence was not available.*"

2006

State of California Report in Response to A.B. 284, Chapter 550, Statutes of 2001

Indoor Mold: A General Guide to Health Effects, Prevention, and Remediation. (CRB-06-001 , January 2006)

Kenneth W. Umbach, Ph.D., and Pamela J. Davis, R.N., P.H.N.

Page 72 "Some experts believe that the ACOEM statement understates risks and effects."

Page 75 "The question of whether health effects result from indoor exposure to mycotoxins is controversial, as stated in the text and is noted above. The conclusion in the present report that such effects are at least plausible reflects, for example ..."There is an accumulated weight of evidence

linking indoor airborne mold and/or mycotoxin exposures to multisystem adverse human health effects."

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2006

Center for Science in the Public Interest

Washington, DC

Integrity in Science Watch -- Week of 3/31/2006

Allergy Journal Authors Failed to Disclose Conflicts of Interest

The prestigious Journal of Allergy and Clinical Immunology (JACI) last month failed to disclose two physicians' roles as insurance company defense experts in their scientific review "The Medical Effects of Mold Exposure," which downplayed risks to human health from household mold. According to court documents obtained by the Center for Science in the Public Interest, Dr. Abba I. Terr, Stanford University School of Medicine, and Dr. Andrew Saxon, University of California at Los Angeles School of Medicine, were paid up to \$600 an hour for testimony in cases brought by homeowners alleging their illnesses were caused by mold. JACI, the journal of the American Academy of Allergy, Asthma and Immunology (AAAAI), requires authors to disclose conflicts of interest to the editor, who then has discretion in publishing them. In a letter to editor Donald Leung, CSPI urged AAAAI to make disclosure mandatory and prevent authors who fail to disclose conflicts of interest from publishing in the journal for three years.

Week of 4/24/06

Allergy Journal Strengthens Conflicts of Interest Disclosure Policy

The Journal of Allergy and Clinical Immunology (JACI), an Elsevier publication, will require greater financial disclosure from authors and automatically publish those disclosures, the editor said. Two mold experts, Dr. Abba Terr and Dr. Andrew Saxon, failed to disclose their roles as defense witnesses in mold exposure liability lawsuits when publishing a review in the journal earlier this year that downplayed the risks from household mold exposure. Editor Donald Leung said future author conflict of interest forms accompanying JACI submissions will now include "specific questions" about expert witnessing and the journal will "ensure that all published manuscripts will carry a conflict of interest statement regarding each author."

Week of 6/5/06

Environmental Journal Retracts Fraudulent Study on Chromium

[Significance: Journal of ACOEM Retracts Fraudulent Study Authored by Expert Defense Witnesses for Usage in Court]

The Journal of Occupational and Environmental Medicine [Journal of ACOEM] will retract a 1997 article on chromium written under the names of two Chinese scientists after a Wall Street Journal investigation revealed that the article was actually drafted and edited by consultants for a major chromium polluter. Chemrisk, founded and directed by Dennis Paustenbach (see <http://www.IntegrityinScience.org/>), purchased in 1995 JianDong Zhang's original data on the link between chromium-6 in drinking water and cancer in Chinese villages. Chemrisk, which had been hired by Pacific Gas and Electric, the California utility company being sued for chromium contamination, then reworked the data to show that Zhang, who objected to the publication, had reversed his conclusion on the chromium-cancer link. The JOEM retraction, signed by editor Dr. Paul Brandt-Rauf, states that the article did not comply with the journal's policy because "financial and intellectual input to the paper by outside parties was not disclosed." Since its publication, the fake article has influenced regulatory decisions on chromium, including being used by a scientific panel for a 2001 report which forced California health officials to revise a recommendation for how much chromium-6 should be allowed in drinking water.

Week of 6/12/06

Top Allergy Journal Will Publish Contributors' Conflicts of Interest

The nation's leading allergy journal now requires authors to publish their ties to industry whenever their articles appear in that journal. The Journal of Allergy and Clinical Immunology, the official scientific journal of the American Academy of Allergy, Asthma and Immunology, recently adopted [new guidelines](#) requiring authors to disclose consultant arrangements, stock or other equity ownership, patent licensing arrangements, and expert witness testimony. Editor-in-Chief Donald Y.M. Leung initiated the policy change after the Center for Science in the Public Interest uncovered the journal's failure to report that a review on the health risk of mold exposure had been authored by two key defense witnesses in mold liability lawsuits. (See Integrity in Science Watch, 3/31 and 4/24)

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### Summary

Many people have been ill with serious mold/mycotoxin induced illnesses. They have been unable to obtain proper medical treatment prior to the time these illnesses have become progressively and irreversibly debilitating. Many physicians and citizens have been falsely told that mold does not cause serious illness, leaving the medical community and public uneducated and unaware of the true danger.

The medical misinformation promoted for the benefit of the defense in mold litigation has stifled and confused the already young field of science. It has fueled contention. The promotion of the concept "not plausible, improbable, junk science" within the medical community and the general public has been a primary cause for the lack of early detection and timely medical treatment.

This in turn, has cost stakeholders with financial interest in the moldy buildings, unnecessary billions. The misinformation, that has retarded proper medical understanding, has also caused a tremendous increase in financial responsibility for stakeholders. Increased health damages sustained equals increased resultant stakeholder liability. .

Mold itself, has not been the crux of the problem. The denial of illness in an attempt to limit liability has directly caused greater illness - and thereby has caused greater liability. The situation has been wastefully self perpetuating. The defense argument of "not plausible, improbable and junk science" has proven to be its own worst enemy.

Dr Jonathan Borak, overseer for the "peer review process" of the ACOEM Mold Statement, summed the matter up best in an email he wrote in 2002:

Email September 8, 2002  
From: Jonathan Borak, Chair of the Scientific Committee, ACOEM  
To: Dean Grove, Past President, ACOEM  
CC: Edward Bernacki, ACOEM President 2002; Barry Eisenberg, Executive Director ACOEM; Tim Key, ACOEM President 2003.

"Dean et al:

I am having quite a challenge in finding an acceptable path for the proposed position paper on mold. Even though a great deal of work has gone in, it seems difficult to satisfy a sufficient spectrum of the College, or at least those concerned enough to voice their views.

I have received several sets of comments that find the current version, much revised, to still be a defense argument. On the other hand, Bryan Hardin and his colleagues are not willing to further dilute the paper. They have done a lot, and I am concerned that we will soon have to either endorse or let go. I do not want to go to the BOD and then be rejected. That would be an important violation of Bryan. I have assured him that if we do not use it he can freely make whatever other uses he might want to make. If we "officially" reject it, then we turn his efforts into garbage. ...."

Garbage it was, based on the Veritox 2000 'review' and provided credibility by the imprimatur of ACOEM. Once the credibility was established by the ACOEM, the garbage was then spread to other purported state of the art, mold review papers.

The unscientific concept that one could take a single review of rodent studies with math applied and determine all human illness from inhaling mycotoxins indoors could never happen, took on a life of its own and grew. It became understood that one could never become seriously ill from inhaling mold indoors.

No one seemed to remember exactly how this concept came to be. They just knew it to be true because they had read it in many authoritative "state of the art" mold review papers.

The lives, health and financial well being of thousands have been forever damaged because of it.

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And that is the Landmark Significance of the Kelly Ruling on April 14, 2006, Sacramento, California, regarding "Risk from inhaled mycotoxins in indoor office and residential environments. Int J Toxicol 2004; 23: 3-10. Robbins CA, Swenson LJ, Hardin BD. (Veritox, 2004).

The courts have found Veritox 2004 is not plausible, improbable and Junk Science.

Maybe NOW we can get this issue out of the courts and into doctors' offices where it belongs. Maybe NOW we can all stop wasting time, lives and money!

Sharon Kramer

BBA Marketing, University of Mississippi and Advocate for Mold Victims
760-822-8026

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