There are so many statements in ACOEM 2011 that are so clearly wrong that one must wonder are these (1) just mis-statements (called honest errors); (2) legal ploys to set up bogus defense approaches in court; or (3) just plain lies. Having testified against any number of ACOEM 2002-spouting defense consultants who have lied on other multiple issues I think that the possibilities (2) and (3) are one and the same.

Don't forget that establishing the legal basis for an argument designed to deceive a judge and jury was the apparent goal of ACOEM 2002. From where I sit, just about every word printed in ACOEM 2002 was designed to mis-lead, though they did make a huge mistake by acknowledging immune mechanisms hidden in the body of the report.

Let's look at a few misleading comments. First, the ploy is to deny the existence of anything other than mycotoxins as the culprits underlying adverse human health effects in water-damaged buildings (WDB). If only mycotoxins are involved then the mycotoxins must be measured if a plaintiff can succeed in litigation. This idea led to the bogus ideas accepted by courts in cases like Geffcken in California and Herzner in Ohio. And of course, if mycotoxins only made people sick, then which mycotoxin was it? This is the specific causation idea, one that is wholly refuted by WHO and GAO. Further if only mycotoxins are involved then are all fungi making toxins? And if some only make toxins, do these fungi always make toxins? Ploy after ploy. Fortunately, this junk science idea is easily rebutted (*please see the Consensus Report of Expert Treating Physicians released by the POA on 7/27/10*).

Then we have the ploy that tries to deceive people into thinking that mycotoxins can only make people sick if they are swallowed (ingestion). How can this absurdity survive? Simple, ignore the massive literature on inhalation as the route of exposure! Use literature on animals eating contaminated grains and hay, as well as aflatoxin (found in peanut butter), making up "aflatoxin equivalents" as if such a thing existed. Made-up science is not science.

Then we have the idea that the illness from WDB is only found is severely immuno-compromised people. That one is really silly.

And if there is illness it couldn't exist since the no one has proved such a thing exists (our group has published prospective acquisition papers covering over 2000 patients and nearly 500 controls). Once again, delete anything published that shows the truth.

Sure, mold allergy exists and that alone is the reason we want to get people out of WDB. Some people actually believe such altruistic statements. Spiders just want flies to have nice comfy silken sheets to sleep on too.

Then the ACOEM 2011 unveils its favorite ploy, the dose response relationship. This is the most common ruse propped up by the toxicology arm of the defense consultants. The problem is not one toxin, then one response; it is one toxin, then a huge, exponential host response. This response is the genesis of the chronic inflammatory response syndrome that WDB patients have. As soon as the defense boys try to invoke the work of *Sir*

Thomas Hill, citing his short talk given in 1965, ask them to explain the role of genetics; amplified inflammatory responses; and cellular immune responses. They will not be able to provide any logical answer.

The biggest offender in ACOEM, the one that makes the whole statement just pure junk, is the made up science.

Take a look at ACOEM 2011 again, this time with the ploys noted.

Adverse Human Health Effects Associated with Molds in the Indoor Environment

Council of Scientific Advisors and approved by the ACOEM Board of Directors on February 14, 2011. This revised statement updates the previous (2002) position statement which was prepared by Bryan D. Hardin, PhD; Bruce J. Kelman, PhD, DABT; and Andrew Saxon, MD; under the auspices of the ACOEM Council on Scientific Affairs. **Ploy: don't identify who is responsible for the authorship.**

In recent years, the growth of molds in home, school, and office environments has been cited as the cause of a wide variety of human ailments and disabilities **(ploy: no references are given for recent years)**. This evidence-based **(ploy: what evidence?)** statement from the American College of Occupational and Environmental Medicine (ACOEM) discusses the current **(ploy: there is nothing new since 2002!)** state of scientific knowledge as to the nature of fungal- (mold-) related illnesses while emphasizing the possible relationships to indoor environments **(ploy: deny there is anything except fungi)**. Food-borne exposures, methods of exposure assessment, and mold remediation procedures are beyond the scope of this paper **(ploy: they sure do talk about ingestion)**.

"Mold" is the common term for multicellular fungi that grow as a mat of intertwined microscopic filaments (hyphae). Many species of fungi live as commensal organisms in or on the surface of the human body. Exposure to molds and other fungi and their spores is unavoidable except when the most stringent of air filtration, isolation, and environmental sanitation measures are observed, e.g., in organ transplant isolation units **(ploy: try to show that WDB are no different from a non-WDB)**.

Molds and other fungi may adversely affect human health through three processes: 1) allergy; 2) infection; or 3) toxicity. It is estimated that about 10% of the population has allergic antibodies to fungal antigens. Only half of these, or 5%, would be expected to show clinical illness. Furthermore, outdoor molds are generally more abundant and important in airway allergic disease than indoor

molds — leaving the latter with an important, but minor overall role in allergic airway disease **(ploy: indoors we will find fungi that couldn't hurt anyone except some allergy and at that the illness is more commonly caused by outdoors fungi)**. Allergic responses are most commonly experienced as allergic asthma or allergic rhinitis ("hay fever";). A rare, but much more serious immune-related condition, hypersensitivity pneumonitis (HP), may follow exposure (usually occupational) to very high concentrations of fungal (and other microbial) proteins **(ploy: deliberately ignore the inflammatory basis of HP).**

Most fungi generally are not pathogenic to healthy humans **(ploy: what does this mean? Those with genetic susceptibility who are in WDB?).** A number of fungi commonly cause superficial infections involving the feet (tinea pedis), groin (tinea cruris), dry body skin (tinea corporis), or nails (tinea onychomycosis). A very limited number of pathogenic fungi — such as Blastomyces, Coccidioides, Cryptococcus, and Histoplasma — infect non-immunocompromised individuals. In contrast, persons with severely impaired immune function, e.g., cancer patients receiving chemotherapy, organ transplant patients receiving immunosuppressive drugs, AIDS patients, and patients with uncontrolled diabetes, are at significant risk for more severe opportunistic fungal infection **(ploy: only those with pre-existing illnesses could possibly be sickened by WDB)**.

Some species of fungi, including some molds, are known to be capable of producing secondary metabolites, or mycotoxins, some of which find a valuable clinical use, e.g., penicillin and cyclosporine **(ploy: ignore all the rest of fungal inflammagens; and ploy: mycotoxins are our friends)**. Serious veterinary and human mycotoxicoses have been documented following ingestion of foods heavily over-grown with molds **(Ploy: ingestion, here it is)**. In agricultural settings, inhalation exposure to high concentrations of mixed organic dusts — which include bacteria, fungi, endotoxins, glucans, and mycotoxins — is associated with organic dust toxic syndrome, an acute febrile illness **(ploy: such is not the case in WDB; ploy: ignore water intrusion, it is the dust)**. Present concern over human exposure to molds in the indoor environment appears to derive from a belief that inhalation exposures to mycotoxins cause numerous and varied, but generally nonspecific, symptoms **(ploy: downplay a robust literature on inhalation and inflammatory effects of such inflammation that they indeed cite in the Rao and Nikulin rat studies)**.

There is scientific evidence that in certain cases, molds and other fungi may adversely affect human health, and mold has been associated with health issues ranging from coughs to asthma to allergic rhinitis **(ploy: only respiratory allergy if anything)**. However, current scientific evidence does not support the existence of a causal relationship between inhaled mycotoxins in the home, school, or office environment and adverse human health effects **(ploy: ignore the thousands of**

patients studied and papers published from 15 countries by not referencing them). An evaluation of the relevant literature follows **(ploy: they take the stance that *only their citations* are relevant. Nonsense).**

Allergy and other hypersensitivity reactions

Allergic and other hypersensitivity responses to indoor molds may be immunoglobulin E (IgE) or immunoglobulin G (IgG) mediated, and both types of response are associated with exposure to indoor molds **(ploy: the vast percentage of WDB patients have normal IgE levels, *see IgE data*).** Uncommon allergic syndromes, allergic bronchopulmonary aspergillosis (ABPA), and allergic fungal sinusitis (AFS), are briefly discussed for completeness, although indoor mold has not been suggested as a particular risk factor in the etiology of either **(ploy: why mention them except to make us look the other way?).**

1. Immediate hypersensitivity: The most common form of hypersensitivity to molds is immediate type hypersensitivity or IgE-mediated "allergy" to fungal proteins. This reactivity can lead to allergic asthma or allergic rhinitis that is triggered by breathing in mold spores or hyphal fragments. Residential or office fungal exposures may be a substantial factor in an individual's allergic airway disease depending on the subject's profile of allergic sensitivity and the levels of indoor exposures. Individuals with this type of mold allergy are "atopic" individuals, i.e., have allergic asthma, allergic rhinitis, or atopic dermatitis and manifest allergic (IgE) antibodies to a wide range of environmental proteins among which molds are only one participant. These individuals generally will have allergic reactivity against other important indoor and outdoor allergens such as animal dander, dust mites, and weed, tree, and grass pollens. Among the fungi, the most important indoor allergenic molds are Penicillium and Aspergillus species **(ploy: the illness is just allergy).**[1] Outdoor molds, e.g., Cladosporium and Alternaria, as well as pollens, can often be found at high levels indoors if there is access for outdoor air (e.g., open windows) **(ploy: if you find Cladosporium it is a bad guy).**

2. About 40% of the population are atopic and express high levels of allergic antibodies to inhalant allergens. Of these, 25%, or 10% of the population, have allergic antibodies to common inhalant molds.[2] Since about half of persons with allergic antibodies will express clinical disease from those antibodies, about 5% of the population is predicted to have, at some time, allergic symptoms from molds. While indoor molds are well-recognized allergens, outdoor molds are more generally important **(repeated).**

3.

A growing body of literature associates a variety of diagnosable respiratory illnesses (asthma, wheezing, cough, phlegm, etc.), particularly in children, with residence in damp or water-damaged homes.[3-5] Studies have documented increased inflammatory mediators in the nasal fluids of persons in damp buildings, but found that mold spores themselves were not responsible for these changes (**Ploy: now look, spores are not a problem**).[6,7] While dampness may indicate potential mold growth, it is also a likely indicator of dust mite infestation and bacterial growth. The relative contribution of each is unknown, but mold, bacteria, bacterial endotoxins, and dust mites can all play a role in the reported spectrum of illnesses **(Ploy: define the mechanism of illness causation please of bacteria, endotoxins and interaction with fungal products).** Their presence can be minimized by control of relative humidity and water intrusion.

2. Hypersensitivity pneumonitis (HP): HP results from exaggeration of the normal IgG immune response against inhaled foreign (fungal or other) proteins and is characterized by: 1) very high serum levels of specific IgG proteins (classically detected in precipitin tests performed as double diffusion tests); and 2) inhalation exposure to very large quantities of fungal (or other) proteins.[8] The resulting interaction between the inhaled fungal proteins and fungal-directed cell mediated and humoral (antibody) immune reactivity leads to an intense local immune reaction recognized as HP (**ploy: ignore innate immunity**). Most cases of HP result from occupational exposures, although cases have also been attributed to pet birds, humidifiers, and heating, ventilating, and air conditioning (HVAC) systems. The predominant organisms in the latter two exposures are thermophilic actinomyces, which are not molds but rather filamentous bacteria that grow at high temperatures (116°F).

   The presence of high levels of a specific antibody — generally demonstrated as the presence of precipitating antibodies — is required to initiate HP, but is not diagnostic of HP.[9] More than half of the people who have occupational exposure to high levels of a specific protein have such precipitin antibodies, but do not have clinical disease.[8] Many laboratories now measure IgG to selected antigens by using solid phase immunoassays, which are easier to perform and more quantitative than precipitin (gel diffusion) assays. However, solid phase IgG levels that are above the reference range do not carry the same discriminatory power as do results of a precipitin test, which requires much greater levels of antibody to be positive. Five percent of the normal population has levels above the reference value for any one tested material. Consequently, a panel of tests (e.g., 10) has a high probability of producing a false-positive result **(Ploy:**

therefore all tests for anything from mold can't be tested for reliably). Screening IgG antibody titers to a host of mold and other antigens is not justified (**ploy: pre-emptive strike against any testing that might be done**), unless there is a reasonable clinical suspicion for HP, and should not be used to screen for mold exposure.[10]

3. Uncommon allergic syndromes: allergic bronchopulmonary aspergillosis (ABPA) and allergic fungal sinusitis (AFS).[11] These conditions are unusual variants of allergic (IgE-mediated) reactions in which fungi actually grow within a person's airway. ABPA is the classic form of this syndrome, which occurs in allergic individuals who generally have airway damage from previous illnesses leading to bronchial irregularities that impair normal drainage, e.g., bronchiectasis.[12, 13] Bronchial disease and old cavitary lung disease are predisposing factors contributing to fungal colonization and the formation of mycetomas. Aspergillus may colonize these areas without invading adjacent tissues. Such fungal colonization is without adverse health consequence (**Ploy: just a little mold doesn't hurt anyone**) unless the subject is allergic to the specific fungus **(Ploy: only allergy stems from WDB exposure)** that has taken up residence, in which case there may be ongoing allergic reactivity to fungal proteins released directly into the body. Specific criteria have been recognized for some time for the diagnosis of ABPA.[14, 15] As fungi other than Aspergillus may cause this condition, the term "allergic broncho-pulmonary mycosis" has been suggested.

   It has more recently become appreciated that a similar process may affect the sinuses — allergic fungal sinusitis (AFS).[16] This condition also presents in subjects who have underlying allergic disease and in whom, because of poor drainage, a fungus colonizes the sinus cavity. Aspergillus and Curvularia are the most common forms **(Ploy: nonsense, we can't ignore so many others yet they do**), although the number of fungal organisms involved continues to increase. As with ABPA, the diagnosis of AFS has specific criteria that should be used to make this diagnosis.[17-19]

Recommendations

- Individuals with allergic airway disease should take steps to minimize their exposure to molds and other airborne allergens, e.g., animal dander, dust mites, and pollens. For these individuals, it is prudent to take feasible steps that reduce exposure to aeroallergens and to remediate sources of indoor mold amplification **(ploy: just ignore all the inflammatory effects of mold amplification)**. Sensitized individuals may need to keep windows closed, remove pets, use dust mite covers, use high-quality vacuum

cleaners, or filter outdoor air intakes to minimize exposures to inhalant allergens (**ploy: the problem is just outdoor allergy**). Humidification over 40% encourages fungal and dust mite growth and should be avoided. Where there is indoor amplification of fungi, removal of the fungal source is a key measure to be undertaken so as to decrease potential for indoor mold allergen exposure (**ploy: it's just allergy**).

- ABPA and AFS are uncommon disorders while exposure is ubiquitous to the fungal organisms involved. There is no evidence to link specific exposures to fungi in home, school, or office settings to the establishment of fungal colonization that leads to ABPA or AFS **(ploy: just remember that there is no evidence that indoor molds hurt people).**

- Once a diagnosis of HP is entertained in an appropriate clinical setting and with appropriate laboratory support, it is important to consider potential sources of inhaled antigen. If evaluation of the occupational environment fails to disclose the source of antigens, exposures in the home, school, or other occupied space should be investigated **(Ploy: investigation for fungal allergens is all you need to do)**. Once identified, the source of the mold or other inhaled foreign antigens should be remediated.

- Appropriate measures should be taken in industrial workplaces to prevent mold growth, e.g., in machining fluids and where stored organic materials are handled such as in agricultural and grain processing facilities. Engineering controls should be used to reduce potentially contaminated aerosol or particulate generation. If engineering controls are inadequate, personal protective equipment may be needed to minimize worker exposures to aerosols and particulate matter **(ploy: personal protection is the key to avoiding illness).**

Infection

An overview of fungi as human pathogens follows. Exposure to molds indoors is generally not a specific risk factor in the etiology of mycoses except under specific circumstances as discussed below for individual types of infection.

1. Serious fungal infections: A very limited number of pathogenic fungi such as Blastomyces, Coccidioides, Cryptococcus, and Histoplasma infect normal subjects and may cause a fatal illness. However, fungal infections in which there is deep tissue invasion are primarily restricted to severely immunocompromised subjects, e.g., patients with hematologic neoplasms including acute leukemia, cancer patients receiving intense chemotherapy, or persons undergoing bone marrow or solid transplantation who receive

potent immunosuppressive drugs.[20] Uncontrolled diabetics and persons with advanced AIDS are also at increased risk. Concern is greatest when patients are necessarily in the hospital during their most severe immunocompromised states, at which time intense measures are taken to avoid fungal, bacterial, and viral infection.[21] Outside the hospital, fungi, including Aspergillus, are so ubiquitous that few recommendations can be made beyond avoidance of known sources of indoor and outdoor amplification, including indoor plants and flowers **(ploy: blame the plants and soil),** because vegetation is a natural fungal growth medium.[22,23]Candida albicans is a ubiquitous commensal organism on humans that becomes an important opportunistic pathogen for immunocompromised subjects. However, it and environmental fungi discussed above that are pathogens in healthy individuals as well (e.g., Cryptococcus associated with bird droppings, Histoplasma associated with bat droppings, Coccidioides endemic in the soil in the southwest U.S.) are not normally found growing in the office or residential environment, although they can gain entry from outdoors. Extensive guidelines for specific immunocompromised states can be found on the Centers for Disease Control and Prevention (CDC) web site at www.cdc.gov.

2. Superficial fungal infections: In contrast to serious internal infections with fungi, superficial fungal infections on the skin or mucosal surfaces are extremely common in normal subjects. These superficial infections include infection of the feet (tinea pedis), nails (tinea onychomycosis), groin (tinea cruris), dry body skin (tinea corporis), and infection of the oral or vaginal mucosa. Some of the common organisms involved, e.g., Trichophyton rubrum, can be found growing as an indoor mold. Others, such as Microsporum canis and T. mentagrophytes, can be found on indoor pets (e.g., dogs, cats, rabbits, and guinea pigs). As a common commensal on human mucosal surfaces, C. albicans can be cultured from more than half of the population that has no evidence of active infection. C. albicans infections are particularly common when the normally resident microbial flora at a mucosal site is removed by antibiotic use. Local factors such as moisture in shoes or boots and in body creases and loss of epithelial integrity are important in the development of superficial fungal infections.

   Pityriasis (Tinea) versicolor is a chronic asymptomatic infection of the most superficial layers of the skin due to Pityriasis ovale (also known as P. orbiculare and Malassesia furfur) manifest by patches of skin with variable pigmentation. This is not a contagious condition and thus is unrelated to exposures, but represents the overgrowth of normal cutaneous fungal flora under favorable conditions.

Recommendations

- Only individuals who are immunocompromised **(ploy: this is the most malevolent false recommendation so far)** need be concerned about the potential for serious opportunistic fungal infections. These individuals should be advised to avoid recognizable fungal reservoirs including, but not limited, to indoor environments where there is uncontrolled mold growth. Outdoor areas contaminated by specific materials such as bird droppings should be avoided as well as nearby indoor locations where those sources may contaminate the intake air **(ploy: blame it on bird poop)**.

- Individuals with M. canis and T. mentagrophytes infections should have their pets checked by a veterinarian. No other recommendations are warranted relative to home, school, or office exposures in patients with superficial fungal infections.

Toxicity

Mycotoxins are "secondary metabolites" of fungi, which is to say mycotoxins are not required for the growth and survival of the fungal species ("toxigenic species") that are capable of producing them. The amount (if any) **(ploy: "if any" is implying that toxins don't always follow indoor mold growth)** and type of mycotoxin produced is dependent on a complex and poorly understood interaction of factors that probably include nutrition, growth substrate, moisture, temperature, maturity of the fungal colony, and competition from other microorganisms.[24-28] Additionally, even under the same conditions of growth, the profile and quantity of mycotoxins produced by toxigenic species can vary widely from one isolate to another.[29-32] Thus, it does not necessarily follow from the mere presence of a toxigenic species that mycotoxins are also present **(ploy: this is one of the most commonly used dodges. See the hundreds of references refuting this deceit is in the POA paper)**.[33-35]
When produced, mycotoxins are found in all parts of the fungal colony, including the hyphae, mycelia, spores, and the substrate on which the colony grows **(ploy: they then ignore the toxins and inflammagens found on fungal fragments, counting spores only)**. Mycotoxins are relatively large molecules that are not significantly volatile[36, 37]; they do not evaporate or "off-gas" into the environment, nor do they migrate through walls or floors independent of a particle **(ploy: presence of microbes in a wall cavity and crawlspace is associated with presence in the respirable air in a building)**. Thus, an inhalation exposure to mycotoxins requires generation of an aerosol of substrate, fungal fragments, or spores **(ploy: if there is a bioaerosol of fragments there will be an exposure to mycotoxins but don't forget that mycotoxins are a very small part of the inflammatory burden found inside WDB).** Spores and fungal fragments do not

pass through the skin, but may cause irritation if there is contact with large amounts of fungi or contaminated substrate material **(ploy: they try to say that dermal contact is a major percentage of exposure at the expense of the contribution of inhalation)** .[38] In contrast, microbial volatile organic compounds (MVOCs) are low molecular weight alcohols, aldehydes, and ketones.[39] Having very low odor thresholds, MVOCs are responsible for the musty, disagreeable odor associated with mold and mildew (**Ploy: the musty smell is usually a fungal product called geosmin**) and they may be responsible for the objectionable taste of spoiled foods.[39,40]

Most descriptions of human and veterinary poisonings from molds involve eating moldy foods **(ploy: most? Says who? This is typical of ACOEM. Allude to non-existent factoids).**[38,40-43] Acute human intoxications have also been attributed to inhalation exposures of agricultural workers to silage or spoiled grain products that contained high concentrations of fungi, bacteria, and organic debris with associated endotoxins, glucans, and mycotoxins.[44,45] Related conditions including "pulmonary mycotoxicosis," "grain fever," and others are referred to more broadly as "organic dust toxic syndrome" (ODTS).[46] Exposures associated with ODTS have been described as a "fog" of particulates[47] or an initial "thick airborne dust" that "worsened until it was no longer possible to see across the room."[48] Total microorganism counts have ranged from 10^5-10^9 per cubic meter of air[49] or even 10^9-10^{10} spores per cubic meter,[50,51] extreme conditions not ordinarily encountered in the indoor home, school, or office environment **(ploy: so what? These authors have no shame trying to imply that non-related exposures are the same. This is just baloney).**

"Sick building syndrome," or "non-specific building-related illness," represents a poorly defined set of symptoms (often sensory) that are attributed to occupancy in a building. Investigation generally finds no specific cause for the complaints **(ploy: actually the cause is exposure to the complex mixture of compounds found in the air and the dust of the building. Here ACOEM 2011 is trying to promote specific causation),** but they may be attributed to fungal growth if it is found. The potential role of building-associated exposure to molds and associated mycotoxins has been investigated, particularly in instances when Stachybotrys chartarum (aka Stachybotrys atra) was identified.[52-55] Critical reviews of the literature[33,56-62] have concluded that indoor airborne levels of microorganisms are only weakly correlated with human disease or building-related symptoms and that a causal relationship has not been established between these complaints and indoor exposures to S. chartarum **(Ploy: note the compound statement here. The second part is the specific causation idea. The first part is trying to get others to agree that monotonic dose response relationships do actually exist regarding immunologic and inflammatory cascades of responses. That idea is nonsense.)**

A 1993-94 series of cases of pulmonary hemorrhage among infants in Cleveland, Ohio, led to an investigation by the CDC and others. No causal factors were suggested initially,[63] but eventually these same investigators proposed that the cause had been exposures in the home to S. chartarum and suggested that very young infants might be unusually vulnerable.[64-66] However, subsequent detailed re-evaluations of the original data by CDC and a panel of experts led to the conclusion that these cases, now called "acute idiopathic pulmonary hemorrhage in infants,"[67] had not been causally linked to S. chartarum exposure.[68] **(ploy: actually one of the authors of ACOEM 2002 boasted in deposition in the Scotia Prince case how he had basically single-handedly undermined the effort of the Cleveland investigators from his role in NIOSH. The Cleveland cohort remains one with a vast murky aspect of the involvement of the CDC with denial of human illness.)**

If mycotoxins are to have human health effects, there must be an actual presence of mycotoxins, a pathway of exposure from source to susceptible person, and absorption of a toxic dose over a sufficiently short period of time (**Ploy: this is a bastardization of Hill. There is no reference here, a sure tip-off to junk science sold as fact. Defense lives and dies on these ideas. Since the detection of mycotoxins is but a mere morsel of what the air of a building has measurement of toxins is irrelevant. Don't be deceived, the pathways are inhalation of bioaerosols with reservoirs in air, dust, possessions and communicating air.**). As previously noted, the presence of mycotoxins cannot be presumed from the mere presence of a toxigenic species **(ploy: they don't stop with the same nonsense rebutted before)**. The pathway of exposure in home, school, and office settings may be either dermal (e.g., direct contact with colonized building materials) **(Ploy: they said "no" earlier!)** or inhalation of aerosolized spores, mycelial fragments, or contaminated substrates **(Ploy: this is right, see how they try to tear down the idea).** Because mycotoxins are not volatile, the airborne pathway requires active generation of that aerosol. For toxicity to result, the concentration and duration of exposure must be sufficient to deliver a toxic dose (**Ploy: look out, here comes the famous math confabulation**). What constitutes a toxic dose for humans is not known at the present time (**ploy: true, it is the entire exposure!),** but some estimates can be made (**ploy: here it is! ACOEM's horrid speculation based on pure garbage assumptions sold as legit science)** that suggest under what circumstances intoxication by the airborne route might be feasible.

Experimental data on the in vivo toxicity of mycotoxins are scant. Frequently cited are the inhalation LC_{50} values determined for mice, rats, and guinea pigs exposed for 10 minutes to T-2 toxin, a trichothecene mycotoxin produced by Fusarium spp.[69, 70] Rats were most sensitive in these studies, but there was no mortality in

rats exposed to 1.0 mg T-2 toxin/m^3. No data were found on T-2 concentrations in Fusarium spores, but another trichothecene, satratoxin H, has been reported at a concentration of 1.0 x 10^{-4} ng/spore in a "highly toxic" S. chartarum strain, s. 72.[29] To provide perspective relative to T-2 toxin, 1.0 mg satratoxin H/m^3 air would require 10^{10} (ten billion) of these s. 72 S. chartarum spores/m^3. **(ploy: they use a single rat study and try to make up all kinds of conclusions about chronic exposure human illness from a one-time animal exposure. The authors of the rat study said that nothing about chronic exposure can be concluded from acute exposure; the criticisms are outlined sequentially in the POA paper. Leaving in this kind of discredited work in 2011 is an example of deliberate contempt of the scientific process.)**

In single-dose in vivo studies, S. chartarum spores have been administered intranasally to mice[29] or intratracheally to rats.[71, 72] High doses (30 x 10^6 spores/kg and higher) produced pulmonary inflammation and hemorrhage in both species. A range of doses was administered in the rat studies and multiple, sensitive indices of effect were monitored, demonstrating a graded dose response with 3 x 10^6 spores/kg being a clear no-effect dose. Airborne S. chartarum spore concentrations that would deliver a comparable dose of spores can be estimated by assuming that all inhaled spores are retained and using standard default values for human subpopulations of particular interest[73] — very small infants, [a] school-age children,[b] and adults.[c] The no-effect dose in rats (3 x 10^6 spores/kg) corresponds to continuous 24-hour exposure to 2.1 x 10^6 spores/m^3 for infants, 6.6 x 10^6 spores/m^3 for a school-age child, or 15.3 x 10^6 spores/m^3 for an adult.

That calculation clearly overestimates risk **(ploy: this calculation has noting to do with risk! It has nothing to do with anything.)** Because it ignores the impact of dose rate by implicitly assuming that the acute toxic effects are the same whether a dose is delivered as a bolus intratracheal instillation or gradually over 24 hours of inhalation exposure. In fact, a cumulative dose delivered over a period of hours, days, or weeks is expected to be less acutely toxic than a bolus dose, which would overwhelm detoxification systems and lung clearance mechanisms. If the no-effect 3 x 10^6 spores/kg intratracheal bolus dose in rats is regarded as a 1-minute administration (3 x 10^6 spores/kg/min), achieving the same dose rate in humans (using the same default assumptions as previously) would require airborne concentrations of 3.0 x 10^9 spores/m^3 for an infant, 9.5 x 10^9 spores/m^3 for a child, or 22.0 x 10^9 spores/m^3 for an adult.

In a repeat-dose study, mice were given intranasal treatments twice weekly for 3 weeks with "highly toxic" s. 72 S. chartarum spores at doses of 4.6 x 10^6 or 4.6 x 10^4 spores/kg (cumulative doses over 3 weeks of 2.8 x 10^7 or 2.8 x 10^5 spores/kg).[74] The higher dose caused severe inflammation with **hemorrhage (ploy: these toxins are benign little chemicals for people, right? So why didn't**

the study animals have assays done that truly reflected what happens in people?), while less severe inflammation but no hemorrhage was seen at the lower dose of s. 72 spores. Using the same assumptions as previously (and again ignoring dose-rate implications), airborne S. chartarum spore concentrations that would deliver the non-hemorrhagic cumulative 3-week dose of 2.8×10^5 spores/kg can be estimated as 9.4×10^3 spores/m^3 for infants, 29.3×10^3 spores/m^3 for a school-age child, and 68.0×10^3 spores/m^3 for adults (assuming exposure for 24 hours per day, 7 days a week, and 100% retention of spores).

The preceding calculations suggest lower bound estimates of airborne S. chartarum spore concentrations corresponding to essentially no-effect acute and subchronic exposures. Those concentrations are not infeasible, but they are improbable and inconsistent with reported spore concentrations. For example, in data from 9,619 indoor air samples from 1,717 buildings, when S. chartarum was detected in indoor air (6% of buildings surveyed) the median airborne concentration was 12 CFU/m^3 (95% CI 12 to 118 CFU/m^3).[75]

Recommendations

- The presence of toxigenic molds within a home, school, or office environment should not by itself be regarded as demonstrating that mycotoxins were present or that occupants of that environment absorbed a toxic dose of mycotoxins **(ploy: here is their litigation mantra)**.

- When mold colonization is discovered in the home, school, or office, it should be remediated after the source of the moisture that supports its growth is identified and eliminated **(ploy: pure lip service. The occupants need to be evaluated by standard published symptom rosters, VCS and labs according to hundreds of patients studied).** Authoritative guidelines for mold remediation are available.[76-78]

- Indoor air samples with contemporaneous outdoor air samples **(ploy: there is no foundation established for this old and dead wrong statement. Outdoors versus indoors is a process that lets people lie.)** can assist in evaluating whether or not there is mold growth indoors; air samples may also assist in evaluating the extent of potential indoor exposure **(ploy: nonsense. No one is suggesting a few air samples can provide useful information to rule out exposure; see GAO, WHO and POA)**. Bulk, wipe, and wall cavity samples may indicate the presence of mold, but do not contribute to characterization of exposures for building occupants **(ploy: there is no foundation for this bogus statement. It of course is not referenced).**

- When patients associate health complaints with mold exposure, treating physicians should evaluate all possible diagnoses, including those unrelated to mold exposure, i.e., consider a complete appropriate differential diagnosis for the patient's complaints. To the extent that signs and symptoms are consistent with immune-mediated disease, immune mechanisms should be investigated **(ploy: this statement is correct. The reality is that ACOEM ignores its own words, providing "death by faint praise."**).

- If a diagnosis of mycotoxicosis is entertained, specific signs and symptoms ascribed to mycotoxins should be consistent with the potential mycotoxins present and their known biological effects at the potential exposure levels involved (**ploy: no one in his scientific mind would suggest that mycotoxins alone are the source of all the diverse inflammatory and cellular immune responses seen in patients with CIRS from WDB**).

Summary

Molds are common and important allergens **(ploy: the summary might be all an attorney will show the jury. The illness isn't allergy).** About 5% of individuals are predicted to have some allergic airway symptoms from molds over their lifetime. However, it should be remembered that molds are not dominant allergens and that the outdoor molds, rather than indoor ones **(ploy: they used this dodge before),** are the most important. For almost all allergic individuals, the reactions will be limited to rhinitis or asthma; sinusitis may occur secondarily due to obstruction. Rarely do sensitized individuals develop uncommon conditions such as ABPA or AFS. To reduce the risk of developing or exacerbating allergies, mold should not be allowed to grow unchecked indoors.

Fungi are rarely significant pathogens for humans. Superficial fungal infections of the skin and nails are relatively common in normal individuals, but those infections are readily treated and generally resolve without complication. Fungal infections of deeper tissues are rare and in general are limited to persons with severely impaired immune systems. The leading pathogenic fungi for persons with non-impaired immune function, Blastomyces, Coccidioides, Cryptococcus, and Histoplasma, may find their way indoors with outdoor air, but normally do not grow or propagate indoors. Due to the ubiquity of fungi in the environment, it is not possible to prevent immune-compromised individuals from being exposed to molds and fungi outside the confines of hospital isolation units **(ploy: these ideas are previously exposed as flawed).**

Some molds that propagate indoors may, under certain conditions, produce mycotoxins that can adversely affect living cells and organisms by a variety of

mechanisms, for example, the ingestion of contaminated foods. Occupational diseases are also recognized in association with inhalation exposure to fungi, bacteria, and other organic matter, usually in industrial or agricultural settings. One mold, Stachybotrys chartarum, is known to be able to produce mycotoxins under appropriate growth conditions. However, years of intensive study have failed to establish exposure to S. chartarum in home, school, or office environments as a cause of adverse human health effects. 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, even for the most vulnerable subpopulations **(ploy: well, here you have it. All the lies are now in one sentence. These factoids are all wrong; none have support in literature and none are accepted by any governmental agency report).**

Mold spores are present in all indoor environments and cannot be eliminated from them. Normal building materials and furnishings provide ample nutrition for many species of molds, but they can grow and amplify indoors only when there is an adequate supply of moisture. Where mold grows indoors there is an inappropriate source of water that must be corrected before remediation of the mold colonization can succeed. Mold growth in the home, school, or office environment should not be tolerated because mold physically destroys the building materials on which it grows, mold growth is unsightly and may produce offensive odors, and mold is likely to sensitize and produce allergic responses in allergic individuals **(ploy: no, mold and all its fellow microbes create a biomixtures that hurts people by causing inflammatory and immunological responses that can be measured, treated, corrected and stabilized to prevent further illness after health is reclaimed).** Except for persons with severely impaired immune systems, indoor mold is not a source of fungal infections. Current scientific evidence does not support the existence of a causal relationship between inhaled mycotoxins in home, school, or office environments and adverse human health effects **(ploy: does current mean only 2002 and before?).**

Acknowledgments

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[a] 5th percentile body weight for 1-month-old male infants, 3.16 kg; respiratory rate for infants under 1 year of age, 4.5 m^3/day.[73]

[b] 50th percentile body weight for 6-year-old boys, 22 kg; respiratory rate for children age 6-9, 10.0 m^3/day.[73]

[c] 50th percentile body weight for men aged 25-34 years, 77.5 kg; respiratory rate for men age 19-65, 15.2 m^3/day.[73]

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