ACOEM 2011 Report Review
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Finally, after many months of extensive review, ACOEM 2011 is here. I hoped to find something in the new version of the artificial paper, ACOEM 2002 that would benefit society. I am sorely disappointed. The sad truth is that there is nothing in the article that would assist the public with understanding the mechanisms of exposure and the possible association with illness. Rather, ACOEM, an entity that purports to exist in the interest of public health, is publishing a statement with the sole purpose of being a document defense consultants can point to as they try to help the insurance industry defeat valid claims of individuals who have become ill following exposure to water-damaged buildings (WDB). When a professional association is making a policy statement, the comments of the organization ideally are meant to help the public in some way, not hurt them. ACOEM 2011 has nothing to help injured persons.

So many people expected that ACOEM 2011 would actually say something scientifically valid, intellectually honest and new. The old report was criticized mercilessly (as well it should have been) as flawed science, dispensed with a lack of thoroughness and lack of transparency. Stated plainly: ACOEM 2002 was nothing more than junk science. As we see, so is ACOEM 2011. There is no room for (1) absence of thoroughness; (2) absence of rigor; and (3) absence of transparency in public documents that will be read by patients and those physicians who are charged with the sacred duty of treating the sick. We swore an oath to do so; we didn't swear an oath to protect assets of insurance companies.

Once again we see the same unreferenced (and wholly incorrect) opinion about chronic exposures: “A cumulative dose delivered over a period of hours, days or weeks is expected to be less acutely toxic than a bolus dose.” Expected by whom? There is no basis in truth to the idea that repeat exposures suppress the subsequent inflammatory response. Actually, just the reverse is true. With re-exposure, “sicker, quicker.”
ACOEM 2011 implies that a monotonic dose response applies to immunologic and inflammatory illness. No, that is just wrong. Yet ACOEM tries to hide behind flawed concepts in toxicology, pointing at mycotoxins as the only components found in WDB that create inflammatory responses in people sickened by exposure to WDB. Wrong, just dead wrong.

Don’t forget that the interior environment of a WDB hosts a complex mixture of inflammmagens, microbes and toxins. Those elements, never identified by spore counting, all elicit an intense innate inflammatory response and a host cellular immune response, particularly in those with genetic susceptibility. We aren’t talking about those few with profound immune suppression here like the bone marrow transplant patient who develops an Aspergillus infection. We are talking about vast numbers of people (1) working, (2) learning or (3) active in their homes who are changed almost overnight by inflammatory responses that explode when neuropeptide regulatory control mechanisms are damaged.

The approach taken by ACOEM 2011 is just the same as what we see from defense interests in mold litigation. They have no data, no research, no human health information on people with ongoing exposure and no data on parameters seen in people with ongoing illness. All they want us to do is to ignore their methods; ignore their desultory approach to the process of decision-making; and ignore their intentional deletion, distortion and misrepresentation of published data.

Remember, ACOEM tries to convince the reader that eating mold spores is the only way to become ill from mold. This idea is based on an illness from moldy hay that affected some starving Russian horses in 1945. To ignore hundreds of documents showing that inhalation, not ingestion, is the mechanism that affects thousands of people is indefensible. But ACOEM plows on. They again try to use a bogus mathematical calculation (ignoring every aspect of well defined inflammatory responses known to occur in affected patients) that they said showed that there couldn’t be enough spores inside any WDB to make anyone sick.

And all this from one acute exposure rat study.

People should not fall for this trap. Look instead at the real science.

I see defense attorneys who still promote ACOEM 2002 as something worthy of consideration for a judge’s opinion in mold litigation. What pure junk science.
You can read the internal emails from 2002 of the ACOEM board as they decided that defense consultants were reasonable people to write a manifesto on mold. Read the emails yourself. Do you think there was fraud here? Was there intentional deception in exchange for something of value?

You can read the exposure of the ACOEM manipulation of science in the Craner paper published in 2008. Add to that the Wall Street Journal piece of 2007 written by David Armstrong that exposed the obvious conflicts of interest of the authors of all the Nay-Sayer papers regarding mold illness. Read the detailed list of reasons why no one should believe ACOEM 2002 published by the Policyholders of America in 7/10 and detailed in Surviving Mold.

We all knew that the members of the ACOEM board who were responsible for acceptance of the 2002 essay had no experience with diagnosis and treatment of human health effects acquired following exposure to WDB. All of us knew they had no basis in experience to write anything about so-called mold illness. Yet ACOEM 2002 survived.

In July 2010, we heard that ACOEM was going to revise their 2002 mold statement. Surely, ACOEM 2011 would have brought us up to date on the nine years of additional laboratory studies and governmental agency publications recognizing the association between WDB, mold exposure and long term illness. But that would not be in the interest of their intended users, those defending the insurance industry. Surely, we thought ACOEM 2011 would accommodate all the new information published in peer reviewed literature (several thousand papers are recorded and annotated in our library) and surely, the ACOEM 2011 would incorporate current US government agency and World Health Organization opinions about human health effects caused by WDB. The passage of time and massive expansion of science would guarantee that any reasonable and objective scientist will review what is known in 2011 for a 2011 consensus statement, right?

Surely, the ACOEM people would solicit opinion from physicians who had first hand knowledge of diagnosis and treatment of mold illness. Surely, ACOEM 2011 would demonstrate a comprehensive and objective review of ALL peer reviewed papers on the ecology and “physiology” of WDB, the potential sources of illness and their interactions, as well as looking at new human health information. That is what we would expect to see.
No, none of the above occurred. Of the 78 references, there are 37 from before 1997; 41 from 1997 to 2002 and none since. NONE. Does the term “intentional deletion” come to your mind as it did mine?

One of the telling criticisms of ACOEM 2002 was the absence of disclosure regarding authorship. ACOEM 2011 provides no attribution of authorship or any evidence that any physician who has published on treatment of human health effects from WDB reviewed this draft. There is no conflict of interest statement either.

Perhaps David Armstrong will return to do another piece on the appearance of deception that flows naturally in the absence of necessary disclosure in medicine.

So now we get to see how ACOEM regards the advances of nine years of science that have helped all governmental agencies. The work of the US GAO (9/08), the WHO (7/09) and the Expert Treating Physicians report to the Policyholders of America (7/10) all expand our understanding of what makes people sick in WDB. But ACOEM 2011 acts like those documents don’t exist!

In the “30,000 foot” view, ACOEM 2011 had the opportunity to correct their earlier wrongs. They did not. The ACOEM 2011 authors could easily have cited work from 2002-2011 to show they were keeping current. They did not. They could have commented on the progress of science, not to mention the opinions in 2011 of the CDC, EPA, WHO and GAO. They did not.

ACOEM 2011 should be retracted. It is an insult to the hard-working members of ACOEM who actually do try to practice good medicine. It is an insult to those sickened by exposure to the interior environment of WDB. It is an insult to science itself.

For more information about ACOEM, see Dr. LaDou’s comments from 2007...

ACOEM 2002 / 2011 Changes

Adverse Human Health Effects Associated with Molds in the Indoor Environment

Council of Scientific Advisors and approved by the ACOEM Board of Directors
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. (Deleted: So-called “toxic mold” has become a prominent topic in the lay press and is increasingly the basis for litigation when individuals, families, or building occupants believe they have been harmed by exposure to indoor molds.) This evidence-based statement from the American College of Occupational and Environmental Medicine (ACOEM) discusses the current state of scientific knowledge as to the nature of fungal-related illnesses while emphasizing the possible relationships to indoor environments. (Deleted: Particular attention is given to the possible health effects of mycotoxins, which give rise to much of the concern and controversy surrounding indoor molds.) Food-borne exposures, methods of exposure assessment, and mold remediation procedures are beyond the scope of this paper. (Incorrect: they talk about ingestion as mechanism of illness acquisition.)

(Deleted: Fungi are eukaryotic unicellular or multicellular organisms that, because they lack chlorophyll, are dependent upon external food sources. Fungi are ubiquitous in all environments and play a vital role in the Earth’s ecology by decomposing organic matter. Familiar fungi include yeasts, rust, smuts, mushrooms, puffballs, and bracket fungi. Many species of fungi live as commensal organism in or on the surface of the human body.) "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., (was for example) in organ transplant isolation units.

Molds and other fungi may adversely affect human health through three processes: 1) allergy; 2) infection; or (was and) 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 (by whom?) 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. 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.

Most fungi generally are not pathogenic to healthy humans. 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.

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. Serious veterinary and human mycotoxicoses have been documented following ingestion of foods heavily overgrown with molds. In agricultural settings, inhalation exposure to high concentrations of mixed organic dusts — which include bacteria, fungi, endotoxins, glucans, and mycotoxins (Just like inside water-damaged buildings! And interiors of wet buildings have much more that is ignored by ACOEM) — is associated with organic dust toxic syndrome, an acute febrile illness. Present concern (this word was “alarm” in 2002) over human exposure to molds in the indoor environment appears to derive (was derives) from a belief that inhalation exposures to mycotoxins cause numerous and varied, but generally nonspecific, symptoms. Deleted here: Current scientific evidence does not support the proposition that human health has been adversely affected by inhaled mycotoxins in the home, school, or office environment.

Added in 2011: 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 (why is
there no reference for this statement?). 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. (New:) An evaluation of the relevant literature follows.

**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. 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.

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. 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). 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. 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. 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. 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.\textsuperscript{6,7} While (was \textit{although}) 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. Their presence can be minimized by control of relative humidity and water intrusion. (Note: \textit{sentence structure changed})

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. 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. 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.\textsuperscript{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.\textsuperscript{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. Screening IgG antibody titers to a host of mold and other antigens is not justified, unless there is a reasonable clinical suspicion for HP, and should not be used to screen for mold exposure.\textsuperscript{10}

3. Uncommon allergic syndromes: allergic bronchopulmonary aspergillosis (ABPA) and allergic fungal sinusitis (AFS).\textsuperscript{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. 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 unless the subject is allergic to the specific fungus 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. 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). 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, 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.

**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. 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. 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.

- 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.
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. 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 (added) may be needed to minimize worker exposures to aerosols and particulate matter (added).

Deleted: Although it is not relevant to indoor mold exposure, it should be mentioned that there is a belief among some health practitioners and members of the public regarding a vague relationship between mold colonization, molds in foods, and a “generalized mold hypersensitivity state.” The condition was originally proposed as the chronic Candida syndrome or Candida hypersensitivity syndrome but now has been generalized to other fungi. Adherents may claim that individuals are colonized with the mold(s) to which they are sensitized and that they react to these endogenous molds as well as to exposures in foods and other materials that contain mold products. The proposed hypersensitivity is determined by the presence of any of a host of non-specific symptoms plus an elevated (or even normal) level of IgG to any of a host of molds. The claim of mold colonization is generally not supported with any evidence, e.g. cultures or biopsies, to demonstrate the actual presence of fungi in or on the subject. Instead, proponents often claim colonization or infection based on the presence of a wide variety of nonspecific symptoms and antibodies detected in serologic tests that represent no more than past exposure to normal environmental fungi. The existence of this disorder is not supported by reliable scientific data.

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., (deleted for example) patients with (deleted lymphoproliferative disorders) hematologic neoplasms including acute leukemia, cancer patients receiving intense chemotherapy, or persons undergoing bone marrow or solid transplantation who receive potent immunosuppressive drugs. 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. 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, because vegetation is a natural fungal growth medium. 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 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.

- 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) 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.\(^{33-35}\)

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. Mycotoxins are relatively large molecules (No, they are some of the smallest biotoxins) 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. Thus, an inhalation exposure to mycotoxins requires
generation of an aerosol of substrate, fungal fragments, or spores. 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. In contrast, microbial volatile organic compounds (MVOCs) are low molecular weight alcohols, aldehydes, and ketones. Having very low odor thresholds, MVOCs are responsible for the musty, disagreeable odor associated with mold and mildew and they may be responsible for the objectionable taste of spoiled foods.

Most descriptions of human and veterinary poisonings from molds involve eating moldy foods. 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. Related conditions including "pulmonary mycotoxicosis," "grain fever," and others are referred to more broadly as "organic dust toxic syndrome" (ODTS). Exposures associated with ODTS have been described as a "fog" of particulates or an initial "thick airborne dust" that "worsened until it was no longer possible to see across the room." Total microorganism counts have ranged from $10^5$-$10^{10}$ per cubic meter of air or even $10^9$-$10^{10}$ spores per cubic meter, extreme conditions not ordinarily encountered in the indoor home, school, or office environment.

"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, 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. Often referred to in the lay press by the evocative, but meaningless terms, "toxic mold" or "fatal fungus," S. chartarum elicits great concern when found in homes, schools, or offices, although it is by no means the only mold found indoors that is capable of producing mycotoxins. Critical reviews of the literature 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.

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, 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. 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," had not been causally linked to S. chartarum exposure.

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. As previously noted, the presence of mycotoxins cannot be presumed from the mere presence of a toxigenic species. The pathway of exposure in home, school, and office settings may be either dermal (e.g., direct contact with colonized building materials) or inhalation of aerosolized spores, mycelial fragments, or contaminated substrates. 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. What constitutes a toxic dose for humans is not known at the present time, but some estimates can be made 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 LC50 values determined for mice, rats, and guinea pigs exposed for 10 minutes to T-2 toxin, a trichothecene mycotoxin produced by Fusarium spp. Rats were most sensitive in these studies, but there was no mortality in rats exposed to 1.0 mg T-2 toxin/m³. 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. To provide perspective relative to T-2 toxin, 1.0 mg satratoxin H/m³ air would require 10¹⁰ (ten billion) of these S. chartarum spores/m³.

In single-dose in vivo studies, S. chartarum spores have been administered intranasally to mice or intratracheally to rats. High doses (30 x 10⁶ 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 (No, no sensitive indices were included at all) were monitored, demonstrating a graded dose response with $3 \times 10^6$ 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 — very small infants, a school-age child, and adults. The no-effect dose in rats ($3 \times 10^6$ spores/kg) corresponds to continuous 24-hour exposure to $2.1 \times 10^5$ spores/m$^3$ for infants, $6.6 \times 10^6$ spores/m$^3$ for a school-age child, or $15.3 \times 10^6$ spores/m$^3$ for an adult.

That calculation clearly overestimates risk 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 $3 \times 10^6$ spores/kg intratracheal bolus dose in rats is regarded as a 1-minute administration ($3 \times 10^6$ spores/kg/min), achieving the same dose rate in humans (using the same default assumptions as previously) would require airborne concentrations of $3.0 \times 10^9$ spores/m$^3$ for an infant, $9.5 \times 10^9$ spores/m$^3$ for a child, or $22.0 \times 10^9$ 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. chartarum spores at doses of $4.6 \times 10^6$ or $4.6 \times 10^4$ spores/kg (cumulative doses over 3 weeks of $2.8 \times 10^7$ or $2.8 \times 10^5$ spores/kg). The higher dose caused severe inflammation with hemorrhage, while less severe inflammation but no hemorrhage was seen at the lower dose of S. chartarum 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 $2.8 \times 10^5$ spores/kg can be estimated as $9.4 \times 10^3$ spores/m$^3$ for infants, $29.3 \times 10^3$ spores/m$^3$ for a school-age child, and $68.0 \times 10^3$ 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.

- (Moved from summary of 2002:) 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. Authoritative guidelines for mold remediation are available.$^{76-78}$

- Indoor air samples with contemporaneous outdoor air samples 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. Bulk, wipe, and wall cavity samples may indicate the presence of mold, but do not contribute to characterization of exposures for building occupants. **Deleted: After the source of moisture that supports mold growth has been eliminated, active mold growth can be eliminated. Colonized porous materials, for example, clothing or upholstery, can be cleaned using appropriate routine methods, e.g., washing or dry cleaning clothing, and need not be discarded unless cleaning fails to restore an acceptable appearance.**

- 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 (**Absolutely! This statement is about the only correct aspect of medical care noted in this statement. The illness is inflammatory and immunologic with nothing to suggest much role for direct toxicity**).

- **Deleted: The possibility of a mycotoxicosis as an explanation for specific signs and symptoms in a residential or general office setting should be entertained only after accepted processes that are recognized to occur have been appropriately excluded and when mold exposure is known to be uncommonly high.** 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.

**Summary**

Molds are common and important allergens. 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, 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.

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 (*reworded*).

*Deleted: Adverse effects of molds and mycotoxins have been recognized for centuries following 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. *Deleted: Molds growing indoors are believed by some to cause building related symptoms despite a voluminous literature on the subject, the causal association remains weak and unproven, particularly with respect to causation by mycotoxins.* 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 (No, this is the most flagrant abuse of science in a litany of scientific abuses).

2002 version of the above: One mold in particular, Stachybotrys chartarum, is blamed for a diverse array of maladies when it is found indoors. Despite its well-known ability to produce mycotoxins under appropriate growth conditions, 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 at best, even for the hypothetically most vulnerable subpopulations.

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. 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.

Acknowledgments

This revised ACOEM position statement was prepared under the auspices of the 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.
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.$^{73}$

References

Note the following references were deleted from the 2011 version:


50. Lacey J, Crook B. Fungal and actinomycete spores as pollutants of the
65. Montaña E, Etzel RA, Allan T, Horgan TE, Dearborn DG. Environmental risk factors associated with pediatric idiopathic pulmonary hemorrhage and
ACOEM: Ploys & Lies
Dr. Ritchie Shoemaker

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 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 inflammannens; 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).

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. 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. 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). 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. 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. 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. 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.

3. Uncommon allergic syndromes: allergic bronchopulmonary aspergillosis (ABPA) and allergic fungal sinusitis (AFS). 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. 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.\textsuperscript{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).\textsuperscript{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.\textsuperscript{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. 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. 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. 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. 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. 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).

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; 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). In contrast, microbial volatile organic compounds (MVOCs) are low molecular weight alcohols, aldehydes, and ketones. 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.\textsuperscript{39,40}

Most descriptions of human and veterinary poisonings from molds involve eating moldy foods (\textit{ploy: most? Says who? This is typical of ACOEM. Allude to non-existent factoids}).\textsuperscript{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.\textsuperscript{44,45} Related conditions including "pulmonary mycotoxicosis," "grain fever," and others are referred to more broadly as "organic dust toxic syndrome" (ODTS).\textsuperscript{46} Exposures associated with ODTS have been described as a "fog" of particulates\textsuperscript{47} or an initial "thick airborne dust" that "worsened until it was no longer possible to see across the room."\textsuperscript{48} Total microorganism counts have ranged from $10^5$-$10^9$ per cubic meter of air\textsuperscript{49} or even $10^9$-$10^{10}$ spores per cubic meter,\textsuperscript{50,51} extreme conditions not ordinarily encountered in the indoor home, school, or office environment (\textit{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 (\textit{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.\textsuperscript{52-55} Critical reviews of the literature\textsuperscript{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 (\textit{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,\textsuperscript{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.\textsuperscript{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,"\textsuperscript{67} had not been causally linked to S. chartarum exposure.\textsuperscript{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 LC50 values determined for mice, rats, and guinea pigs exposed for 10 minutes to T-2 toxin, a trichothecene mycotoxin produced by Fusarium spp.\textsuperscript{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 $1.0 \times 10^{-4}$ ng/spore in a "highly toxic" S. chartarum strain. 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. 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 or intratracheally to rats. High doses ($30 \times 10^6$ 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 $3 \times 10^6$ 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 — very small infants, school-age children, and adults. The no-effect dose in rats ($3 \times 10^6$ spores/kg) corresponds to continuous 24-hour exposure to $2.1 \times 10^6$ spores/m$^3$ for infants, $6.6 \times 10^6$ spores/m$^3$ for a school-age child, or $15.3 \times 10^6$ 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 ($3 \times 10^6$ spores/kg/min), achieving the same dose rate in humans (using the same default assumptions as previously) would require airborne concentrations of $3.0 \times 10^9$ spores/m$^3$ for an infant, $9.5 \times 10^9$ spores/m$^3$ for a child, or $22.0 \times 10^9$ 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. chartarum spores at doses of $4.6 \times 10^6$ or $4.6 \times 10^4$ spores/kg (cumulative doses over 3 weeks of $2.8 \times 10^7$ or $2.8 \times 10^5$ spores/kg). 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. chartarum 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 $2.8 \times 10^5$ spores/kg can be estimated as $9.4 \times 10^3$ spores/m$^3$ for infants, $29.3 \times 10^3$ spores/m$^3$ for a school-age child, and $68.0 \times 10^3$ 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$^3$).

**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.

- 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

This revised ACOEM position statement was prepared under the auspices of the 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: where is the conflict of interest statement? Who wrote the trivial changes made in ACOEM 2002?)

References (ploy: do not expect any rigor, transparency or thoroughness here)


33. Tobin RS, Baranowski E, Gilman AP, Kuiper-Goodman T, Miller JD,
64. Centers for Disease Control and Prevention (CDC). Update: pulmonary
hemorrhage/hemosiderosis among infants — Cleveland, Ohio, 1993-1996.
IOM April 2011

Does the Institute of Medicine writing in April 2011 make comments about consensus statements that would help us evaluate ACOEM and AAAAI 2006? They sure do and the evaluation isn't pretty.

Clinical Practice Guidelines

We Can Trust

Committee on Standards for Developing
Trustworthy Clinical Practice Guidelines
Board on Health Care Services

Robin Graham, Michelle Mancher, Dianne Miller Wolman,
Sheldon Greenfield, and Earl Steinberg, Editors
Foreword

First Paragraph: Clinical Practice Guidelines (CPGs) are intended to provide a systematic aid to making such complex medical decisions. When rigorously developed using a transparent process that combines scientific evidence, clinician experiential knowledge and patient values, CPGs have the potential to improve many clinician and patient healthcare decisions, and enhance healthcare quality and outcomes.

Second Paragraph: The proposed standards cover a number of elements essential to developing sound practice guidelines, including: transparency; conflict of interest; guideline development group composition; CPG-SR intersection; establishing evidence foundations for and strength of recommendations; articulation of recommendations; external review; and updating.
Preface

Paragraph 3: At the same time, there has been considerable concern expressed by physicians, consumer groups, and other stakeholders about the quality of the processes supporting development of CPGs and the resulting questionable validity of many CPGs and CPG-based clinical performance measures. Specifically, this concern extends from limitations in the scientific evidence base on which CPGs rely; a lack of transparency of development groups’ methodologies; conflict of interest among guideline development group members and funders; and questions regarding how to reconcile conflicting guidelines.

Paragraph 4: There was a pressing need for standards regarding many dimensions of guideline development, including the potential for conflict of interest; the importance of transparency of the guideline development process; the appropriate type and level of patient and public input into the CPG development process; the need for clarity regarding the reasoning supporting each CPG recommendation; the approaches used to rate the quality of evidence underlying and strength of each CPG recommendation; the need to ensure that CPGs take account of patients with coexisting conditions; and the relationship between individuals who develop a guideline and those who perform SRs on topics relevant to the CPG.

Summary

Pg. 1 Paragraph 4: Certain factors commonly undermine the quality and trustworthiness of CPGs. These include: variable quality of individual scientific studies; limitations in systematic reviews (SRs) upon which CPGs are based; lack of transparency of development groups’ methodologies (particularly with respect to evidence quality and strength of recommendation appraisals); failure to convene multi-stakeholder, multidisciplinary guideline development groups and corresponding non-reconciliation of conflicting guidelines; unmanaged conflicts of interest (COI); and overall failure to use rigorous methodologies in CPG development.

Summary

Pg. 3 Paragraph 2: Clinical practice guidelines are statements that include recommendations intended to optimize patient care that are informed by a systematic review of evidence and an assessment of the benefits and harms of alternative care options.

To be trustworthy, guidelines should:

- Be based on systematic review of the existing evidence;
- Be developed by a knowledgeable, multidisciplinary panel of experts and representatives from key affected groups;
- Consider important patient subgroups and patient preferences, as appropriate;
• Be based on an explicit and transparent process that minimizes distortions, biases, and conflicts of interest;
• Provide a clear explanation of the logical relationships between alternative care options and health outcomes, and provide ratings of both the quality of evidence and the strength of the recommendations; and
• Be reconsidered and revised as appropriate when important new evidence warrants modifications of recommendations.

Pg. 5 External Review

7.1 External reviewers should comprise a full spectrum of relevant stakeholders, including scientific and clinical experts, organizations (e.g., health care, specialty societies), agencies (e.g., federal government), patients, and representatives of the public.

7.3 The GCG should consider all external reviewer comments and keep a written record of the rationale for modifying or not modifying a CPG in response to reviewers’ comments.

Summary: Conclusion

Pg. 9: Clinical decisions are made under uncertainty. Yet, as medical, biomedical, and health services research advance, translation of scientific evidence increasingly reduces uncertainty in clinical practice. However, requisite to this promise are clinician and patient access to trustworthy clinical practice guidelines informed by high-quality evidence and a guideline development process reflective of best practices. The committee believes the eight standards proposed herein, when embraced by guideline developers, have the capacity to increase quality and trustworthiness of CPGs and ultimately enhance healthcare quality and patient outcomes.

Introduction Chapter 1

Pg. 14: Development standards that the committee identified included

• Objectivity in both the development process and the conclusions, resulting from a balanced panel;
• Transparency of the deliberative process in all aspects, including conflicts of interest among panelists or the sponsoring organization, the methods of data gathering and evidence assessment, and evaluation of strength of recommendations;
• Efficiency and timeliness when considering clinicians’ need for timely advice and minimization of duplication and resource wastage by guideline developers;
• External review by outside experts of draft guidelines and independent party oversight of developer responses to reviewer comments;
• Continuous monitoring of relevant literature so that guidelines are reevaluated when important new evidence is produced and their currency is ensured; and
• Reduction of overlaps and gaps through voluntary efforts to increase consensus among various organizations developing guidelines on the same topics and to address topics that need guidelines (IOM, 2008)

Page 14: Many current clinical practice guidelines suffer from limitations in the scientific evidence base and shortcomings in the guideline development process (IOM, 2008; Shaneyfelf and Centor, 2009). First, the evidence base is limited by an absence or paucity of studies and systematic reviews of the efficacy and effectiveness of medical and surgical procedures, treatments, drugs, and devices. Consequently, CPG recommendations often rely on low quality evidence or expert opinion.

Pg. 15: The quality of many CPGs is further diminished by the process used to develop some CPGs, including: CPG development panel formation without sufficient attention to conflicts of interest (either financial or intellectual) or to balancing bias and including all relevant topical and methodological disciplines and stakeholders; poor coordination with systematic review groups, which does not permit tailoring of reviews to the specific CPG topic; and a lack of transparency concerning the derivation of recommendations (Coates, 2010; Jacobs, 2010; Koster, 2010). GDGs may cherry pick studies that support their positions, even when high quality SRs are available.

Page 18: PURPOSE AND UPDATED DEFINITION OF CLINICAL PRACTICE GUIDELINES

Clinical Practice Guidelines are statements that include recommendations intended to optimize patient care. They are informed by a systematic review of evidence and an assessment of the benefits and harms of alternative care options. To be trustworthy, guidelines should

• Be based on a systematic review of the existing evidence;
• Be developed by a knowledgeable, multidisciplinary panel of experts and representatives from key affected groups;
• Consider important patient subgroups and patient preferences, as appropriate; conflicts of interest;
• Provide a clear explanation of the logical relationships between alternative care options and health outcomes, and provide ratings of both the quality of evidence and the strength of recommendations; and
• Be reconsidered and revised as appropriate when important new evidence warrants modification of recommendations.

Conflict of Interest

Page: 44. Conflict of interest among guideline developers continues to be a worrisome area for guideline users. Public forum testimony to the committee indicated that COI is particularly concerning to many types of stakeholders.
Chapter 4 Establishing Transparency

Page 57: “Transparency” connotes the provision of the information to CPG users that enables them to understand how recommendations were derived and who developed them. Increasing transparency of the guideline development process has long been recommended by authors of CPG development appraisal tools.

Pg. 59: Disclosure policies should relate to all potential committee members (including public/patient representatives) and should include all current and planned financial and institutional conflicts of interest. Financial (commercial or non-commercial) COI typically stems from actual or potential direct financial benefit related to topics discussed or products recommended in guidelines. Direct financial commercial activities include clinical services from which a committee member derives a substantial proportion of his or her income; consulting, board membership for which compensation of any type is received; servings as a paid expert witness; industry-sponsored research; awarded or pending patents; royalties; stock ownership or options; and other personal and family member financial interest. Examples of non-commercial financial activities profit organizations (Schunneman et al., 2009). A person whose work or professional group fundamentally is jeopardized, or enhanced, by a guideline recommendation is said to have intellectual COI. Intellectual COI includes authoring a publication or acting as an investigator on a peer-reviewed grant directly related to recommendations under consideration. Finally, individuals with knowledge of relationships between their institutions and commercial entities with interests in the CPG topic are considered to have institutional COI. These include public/patient representative from advocacy organizations receiving direct industry funding.

Source:

http://www.survivingmold.com/legal-resources/publications/acoem