Clinical Reviews in Allergy & Immunology (2019) 57:449–455
https://doi.org/10.1007/s12016-019-08767-4

The Myth of Mycotoxins and Mold Injury

Christopher Chang [1,2] · M. Eric Gershwin [1]

Published online: 13 October 2019

Abstract
In recent years, mold has been blamed for many symptoms or a constellation of symptoms. These symptoms are usually vague and subjective and difficult or impossible to measure or quantify. Moreover, there is no scientific evidence that mold has anything to do with these symptoms. In particular, the concept of toxic mold syndrome has permeated the public consciousness, and mycotoxins have falsely been associated with autoimmune diseases and a variety of other conditions. In fact, there is no evidence that the presence of mycotoxins in the air is enough to cause any disease known to man. Molds legitimately can cause allergies and can be a trigger for asthma. Certain specific molds such as *Aspergillus* can be a cause of hypersensitivity pneumonitis. In immunocompromised hosts, both dermatologic and systemic infections can result from various fungi and can be associated with significant morbidity or even mortality. However, the existence of toxic mold syndrome has been disproven, despite the numerous disreputable practices such as testing homes for mold spores, measuring "mycotoxins" in the urine, and testing patients for IgG to mold. In truth, none of these techniques have been validated, nor do they have any relevance to any clinical disease. All that these tests that are being performed by laboratories of disrepute does is to further propagate misinformation and inflict unnecessary and often exorbitant costs on patients desperate for a clinical diagnosis, right or wrong, for their constellation of maladies.

Keywords Mycotoxins · Mold spores · Allergic rhinitis · Allergic fungal sinusitis · Allergic bronchopulmonary aspergillosis · Stachybotrys · Toxic black mold

Introduction

In the 1990s, a report emerged of six infants with pulmonary hemorrhage resulting from exposure to mycotoxins [1]. At the time, a great deal of attention was given to a condition called "sick building syndrome." Mass hysteria ensued, with numerous people complaining of vague symptoms such as headache, difficulty concentrating, loss of memory, fatigue, myalgia, and mood changes. Various factors were being proposed as culprits, including formaldehyde, volatile organic compounds, mold spores, and mycotoxins. A disease named "toxic mold syndrome" or "toxic black mold" was invented to explain many of these cases. Ultimately, despite multiple studies, there was no confirmation that any living or non-living component of a building could possibly be the cause of these subjective and vague symptoms. Even the early reports of an association between pulmonary hemorrhage and mycotoxins was refuted. [2] In addition, there was no study that could provide any evidence of any logical pathogenesis that could explain how exposure to mold could cause these conditions.

Even in the face of a lack of such evidence, toxic mold syndrome took on a life of its own, leading to multiple cases of vague symptoms and "mold-related illness" being tried in a courtroom, with tenants suing landlords or building managers for their illness, and "expert" witnesses were retained on both sides. Enormous amounts of legal fees were being spent on cases that relied on this junk science. This went on for years during the early 2000s, but eventually, because of a lack of scientific evidence, the hype has been tempered but not extinguished. The media fueled this junk science and to this day, you can go to the internet and find search results that read "TOXIC MOLD CAN MAKE YOU SICK!" Now, most scientists and clinicians have accepted that there is a lack of evidence for "toxic mold syndrome." Of course, there are bastions of resistance,

✉ Christopher Chang
chrchang@mhs.net

✉ M. Eric Gershwin
megershwin@ucdavis.edu

[1] Division of Rheumatology, Allergy and Clinical Immunology, University of California at Davis, Davis, CA 95616, USA

[2] Division of Pediatric Immunology and Allergy, Joe DiMaggio Children's Hospital, Hollywood, FL 33021, USA

and every now and then, cases of mold-related illness are still reported in the literature, and like many other such falsehoods, some members of the public and sadly, of the medical profession, remain convinced of its existence.

Mold-Associated Conditions or Diseases

Allergic Diseases

A practicing allergist will often encounter patients who believe that they suffer from mold-related diseases, whether that may be allergies or otherwise. This is because mold growth results from water issues, either water leaks or high humidity within an enclosed structure, and when mold grows, it is visible. When a patient sees gray, brown, or black stains on sheetrock or plasterboard, and they develop symptoms consistent with an allergic disease such as allergic rhinitis or asthma, it is easy for mold to be blamed. Unfortunately, this belief is often reinforced by other non-professional or even professional people in whom they may confide. In truth, with a few exceptions, there has been no evidence that visible mold correlates with fungal spores in the air. In addition, in many of these cases, the symptoms do not match the disease. In other words, if someone presents with headache, it is unlikely to be allergic in nature. Headache is simply not a symptom of allergy. In the patient truly suffering from allergic rhinitis, differential diagnosis includes chronic rhinitis, non-allergic rhinitis with eosinophilia (NARES), anatomical issues, cholinergic or gustatory rhinitis, and many other conditions. If the patient is complaining of headache or vague symptomatology such as loss of concentration or memory loss, then allergic diseases themselves should not even be in the differential diagnosis.

Centuries ago, people suffering from allergies were described as having "Rose Catarrh," and their symptoms were blamed on the most visible and colorful flower around, namely roses. But as we learned more about allergies, we began to understand that pollen is transported either by insects or wind, and it is wind-pollinated plants that confer the worst allergies. It turns out that roses are typically insect pollinated and could not be the culprit. Similarly, just because there is visible mold does not mean that this is what is causing disease. In fact, even if there is visible mold, if a patient has a negative skin prick or specific IgE blood testing to mold, then it is likely their symptoms are not due to mold, but another allergen, most probably dust mites. To just assume that a patient is mold allergic or suffers from any type of mold-related disease just because there is mold in the home is profoundly unscientific. Even if the patient has a positive skin test or specific IgE test for mold and has allergy symptoms does not mean that the patient has an allergy to mold, since both in vivo and in vitro tests only test for sensitization and not clinical allergy. Dust mites and pet dander, which often co-exist in homes where there is mold, are actually much more potent than mold spores as allergenic proteins and are much more relevant than mold spores in patients with allergic rhinoconjunctivitis or asthma.

On the other hand, if a patient truly is sensitized to mold, and suffers from nasal symptoms such as rhinorrhea, congestion, or sneezing, then the mold spores can be a cause of their allergies. The pathophysiology of allergic rhinitis is through cross-linking of antigen-specific IgE on the surface of mast cells leading to release of mediators such as histamine and other inflammatory substances. Some of these are preformed and can be released rapidly, thus the immediate type response experienced by sufferers of allergic rhinitis and allergic conjunctivitis.

Avoidance is always the first-line treatment of any allergy. In some cases, it is possible and others not. In the case of mold, the best treatment would be to eradicate the mold, and there are various methods to achieve this, including removing the source of moisture, replacing or remodeling the damaged parts of the home, or killing the mold with bleach. Treatment with medications such as antihistamines and intranasal steroids may be helpful. Mold immunotherapy to desensitize the patient to the molds to which they are sensitized is another option, but mold immunotherapy typically does not work as well as for other allergens such as pollens, dust mites, or pet dander.

It should also be stated that measuring IgG to molds serves no purpose in the diagnosis of mold allergy. It is common for patients to present with either valid or non-valid symptoms of allergy, armed with blood test results of IgG levels to various molds, as well as an inspection report of the home from some mold spore company that is some 50 pages long, and puts the fear of death into patients because these reports essentially inform the patient that any or all the mold spores that they have detected is going to kill them. A conflict of interest exists as well, as the companies that perform the testing are usually the companies involved in the remediation. Typically, the patients have wasted a large sum of their hard-earned money on both the blood testing (which is often ordered by physicians with no knowledge of allergic diseases, or may not even be ordered by a physician but is performed by irreputable companies that reach out directly to patients), and on the inspection of their homes. Measurement of mold concentrations on tape lift specimens are flawed and do not correlated with fungal exposure in the breathing zone. Another consideration is if the patient only has indoor symptoms, but the levels of mold spores as measured in the air inside the house is similar to that outside, then mold-related allergy can be ruled out. It should also be noted that mold sampling reveals highly variable results over time. [3] Moreover, the CDC neither recommends environmental testing for molds nor evaluation of patients for mycotoxin-related disease, even in those who work or live in water-damaged buildings, in part because of the lack of clinical relevance of these tests (see below under mycotoxins). [4, 5]

Hypersensitivity Pneumonitis

There are a number of hypersensitivity pneumonitis syndromes in which molds have been found to be the culprit. The most common hypersensitivity pneumonitis is allergic bronchopulmonary aspergillosis. Allergic fungal sinusitis is also a type of hypersensitivity pneumonitis. Although the isotype of antibody found against the mold species commonly attributed to these diseases is unclear, it has been found that in these cases, IgG may play a role in pathogenesis. It should be noted that the molecular pathogenesis of hypersensitivity pneumonitis is still a mystery. Nonetheless, the diagnosis of both ABPA and AFS is not made by the presence or absence of IgG or IgE to *Aspergillus* and other molds alone, but by clinical history, skin testing, specific IgE testing, serum IgE levels, and imaging studies (Table 1).

Other forms of hypersensitivity pneumonitis with a mold etiology include cheese-washer's lung, compost lung, farmer's lung, humidifier lung, Japanese summer house AP, malt worker's lung, lycoperdonosis, sauna worker's lung, sequoiosis, wine grower's lung, and woodworker's lung. As can be inferred by the descriptive names of these conditions, they occur in occupational settings where extremely high exposures are encountered. Furthermore, with each condition, there are specific (often only one) fungal species that is associated with the disease (Table 2), which means that ordering large panels of mold sensitivities in patients whose occupation may predispose them to disease is simply a fishing expedition of no value. Moreover, as the name of each condition suggests, these mostly affect the lung, so that symptoms which are vague, subjective, and impossible to measure have no place in the diagnosis of these conditions. If it is deemed that symptoms are potentially consistent with hypersensitivity pneumonitis, then the differential diagnosis of these symptoms (cough, fever, dyspnea, but not other vague subjective symptoms) would include various forms of interstitial lung diseases, such as usual interstitial pneumonia, nonspecific interstitial pneumonia, lymphocytic interstitial pneumonia, respiratory bronchiolitis associated with interstitial lung disease, follicular bronchiolitis, Langerhans cell histiocytosis, and sarcoidosis.

Table 1 Criteria for the diagnosis of ABPA

1. Predisposing conditions[1]
   a. Asthma
   b. Cystic fibrosis
2. Obligatory criteria[2]
   a. Aspergillus skin test positivity or positive IgE serum antibodies to *Aspergillus fumigatus*
   b. Elevated serum IgE greater than 1000 IU/ml[3]
3. Other criteria[4]
   a. Precipitating serum antibodies to *Aspergillus fumigatus*
   b. Total eosinophil count greater than 500 cells per μl, in glucocorticoid naïve patients
   c. Radiographic pulmonary nodules consistent with ABPA

[1] One of these conditions must exist

[2] Both must be present

[3] If all other criteria are met, a level less than 1000 IU/ml is accepted

[4] Must have at least two of these criteria

Infectious Diseases

Fungi can also cause infections. Tinea corporis, tinea capitas, tinea pedis, and the other tineas are examples of dermatologic fungal infections that affect the skin and nails of various parts of the body, with the body part indicated by their name (e.g., tinea pedis is fungal infection of the skin of the feet). [6] These infections are primarily caused by *Trichophyton*, *Microsporum*, and *Epidermophyton* and are not caused by exposure to airborne mold spores. Systemic or organ-specific fungal infections are a real disease, and they occur in mostly immunocompromised hosts. [7–9] *Candida* causes thrush or diaper rashes or even systemic disease in infants and immunocompromised individuals. [10] But these conditions are diseases with clear physical findings, not subjective complaints raised by those people who claim to have a nebulous condition called "systemic candidiasis." Most of the latter patients complain of the same vague symptomatology that sick building syndrome patients or toxic mold syndrome patients do. Systemic fungal infections can occur in immunocompromised hosts, but the signs and symptoms are clear, well defined, and objective, such as fever, abnormalities on imaging, positive cultures, or positive serology.

Diseases with no Scientific Basis or Plausible Pathogenesis

Toxic Mold Syndrome

Stemming from the reports of pulmonary hemorrhage in infants in Cleveland in 1994 was the concept that airborne toxins released by molds can cause serious physiological diseases, primarily of the lung. The media coverage that ensued further encouraged the belief even in the absence of studies confirming these associations. Symptoms were non-specific and included a variety of respiratory symptoms such as fatigue or headache. The term sick building syndrome became fashionable at the time, as mass hysteria of building occupants followed the appearance of one or a few people who developed vague symptoms of unknown etiology. The primary species blamed for the release of mycotoxins was *Stachybotrys chartarum*. But only about a third of the strains of *Stachybotrys* produce mycotoxins. The mycotoxins include the macrocyclic trichothecenes, satratoxins G and H. [11, 12] When these chemicals are detected in air, the amounts are widely variable, but typically have been in the range of 0.25 to 0.43 ng/m^3 in a water-

Table 2 Hypersensitivity pneumonitis conditions with a mold etiology

Condition	Exposure	Specific trigger
Cheese washer's lung	Cheese casings	*Penicillium casie*, *Penicillium rogueforti*
Compost lung	Compost	*Aspergillus*
Farmer's lung	Moldy hay	*Aspergillus*, *Saccharopolyspora rectivirgula*
Humidifier lung	Humidifiers	*Aureobasidium pullulans*
Japanese summer house HP	Damp wood, mats	*Trichosporon cutaneum*
Lycoperdonosis	Puffballs	*Puffball spores*
Malt worker's lung	Moldy barley	*Aspergillus clavatus*
Maple bark disease	Moldy maple bark	*Cryptostroma corticale*
Sauna worker's lung	Contaminated water	*Aureobasidium*, *Graphium*
Sequoiosis	Redwood tree bark	*Aureobasidium*, *Graphium*
Tobacco worker's lung	Moldy tobacco	*Aspergillus*
Wine grower's lung	Moldy grapes	*Botrytis cinerea*
Woodworker's lung	Moldy wood, dust	*Alternaria*, *Penicillium*

damaged building. In fact, dust sampled for mycotoxins have shown less than 1–43 pg/mg of dust. At these concentrations, an individual in such an environment breathing normally for 8 h would only inhale 0.72 and 1.2 ng of satratoxins G and H, respectively, far less than the amount humans are exposed to in their normal daily activities. [13–15]

Mycotoxins that have been studied in animals and implicated in human health and disease include trichothecenes, aflatoxin, zearalenone, citrinin, fumonisins, ergot alkaloids, ochratoxin A, and patulin. A recent review follows the mode of operation of most paper on mycotoxicosis—they begin with the premise that mycotoxins are harmful, without critically evaluating the evidence. According to the authors, researchers can barely agree upon a definition or a classification of mycotoxins. In their assessment, they concede that the field is rampant with studies of questionable scientific merit. Mycotoxins in children have not been well studied, and true clinical meaningful associations between mycotoxin and the health of children are scarce. [16] In fact, the great majority of original research papers on mycotoxins is done in conjunction with the farm animal industry.

Another area of active research is the impact of mycotoxins on the gut microbiome. [17] A recent paper evaluated mycotoxins as a possible link to "intestinal health." But even these studies admit that the relationship is strictly hypothetical. [18] More importantly, in a literature search on mycotoxins, the majority of papers were related to animal health and not human health. Many of the conclusions are drawn from extrapolation of these studies to humans, and the studies that were done in humans were primarily published in journals that were created to promulgate the idea that mycotoxin exposure in the doses that normal humans experience is hazardous to human health. Other issues that plague these studies are poor study design, lack of randomization or a control group, lack of clinical relevance or significance, and overstating the conclusions.

Mycotoxins have also been blamed for autism. However, a recent paper showed no association between autism and mycotoxin exposure in school-aged children. [19] In recent years, a number of disreputable laboratories have been adapting the methodology for testing mycotoxins in pig urine to human patients, in an attempt to link mycotoxins in the urine with a variety of diseases, including autoimmune diseases, or to explain the vague symptoms reported by these patients. These labs will perform these tests in the absence of a physician's order and will charge exorbitant fees to the patient. The result of these types of unfounded testing is the propagation of misinformation and the needless cost to the patient, for a test that has not clinical significance whatsoever. In fact, there are no FDA-approved urine tests for mycotoxins [20], and there are normally found low levels of so-called "mycotoxins" in foods. [21, 22] Often, patients who present with a diagnosis of "mycotoxicosis" are treated with anti-fungal medications, which is ludicrous and illogical because these medications are designed and indicated for the treatment of fungal infections (see above), and not the effects of toxins. [23]

Mold and Autoimmunity

There is no evidence for any relationship between mold exposure and autoimmune diseases. It is known that the pathogenesis of autoimmune diseases is probably multifactorial, with both genetic and environmental factors playing a role. In addition, epigenetic modifications can be triggered by environmental exposures to cause aberrant expression of genes and induce disease. Recently, the microbiome has been studied as a determining environmental factor for the development of autoimmunity, but molds have not been specifically studied.

Chronic autoimmune demyelinating polyneuropathy (CIDP) is thought to be an autoimmune disease characterized by symmetric motor weakness. While it appears that there is activation of cellular immunity based on the activation of T cells and the deposit of immunoglobulin and complement on myelinated nerve fibers, the pathogenesis is still unknown,

and the use of the term "autoimmune" in its name is assumed, not proven. As in many other conditions with subjective symptoms and poorly defined criteria, patients are commonly over- or misdiagnosed. [24] As in most autoimmune diseases, the pathogenesis is most likely multifactorial, with both genetic and environmental factors lining up in an unfavorable pattern that results in autoimmunity. [25] On the other hand, while it may be popular today to blame CIDP on mold or mycotoxin exposure, there has never been any scientific evidence to implicate mold in any autoimmune disease, nor has there been any plausible pathogenesis that involves mold exposure.

Table 3 Examples of junk science—conditions, treatments, associations of dubious scientific merit

Illness, association, or treatment	Description	Reference
Toxic mold syndrome	An attempt to associate mycotoxins with a variety of vague and subjective symptoms has led to numerous media stories about toxic black mold. There is no scientific evidence to back this up, nor is there any plausible mechanism by which this can occur	[32]
Sick building syndrome	Invented at about the same time as toxic mold syndrome, this condition also attempts to link headaches, fatigue, myalgias, and a myriad of subjective and vague symptoms with building components, including volatile organic compounds and formaldehyde. No solid scientific evidence has been found to support these claims	[33]
Vaccines and autism	The anti-vaxers have adopted Andrew Wakefield's 1998 fraudulent study in which he fabricated data to match his theory that the MMR vaccine can cause autism. There is not a shred of evidence that this occurs, but it has developed into a popular culture that threatens the health of all children and adults, including those who legitimately cannot receive vaccines for medical reasons	[34]
PANDAS	An acronym for pediatric autoimmune neuropsychiatric disorders associated with Streptococcus, this disorder attempts to associated changes in behavior and movement to a prior Streptococcal infection, without any plausible pathophysiology or mechanism for why this could conceivably occur. In fact, the autoimmune paradigm in PANDAS has yet to be proven	[35]
CRPS	Complex regional pain syndrome. Pain specialists gathered together to create this entity and to give patients with vague pain symptoms a label. It is a label that has contributed to the opioid epidemic as this legitimized drug seeking behavior	[36]
Chronic fatigue syndrome	Also known as fibromyalgia, patients with vague symptoms of fatigue and myalgias now have a name to their condition. The pathophysiology is unknown	[37]
Homeopathy	A form of therapy in which miniscule amounts of usually harmless molecules is used to treat a myriad of conditions. Homeopathy is not supported by scientific evidence and the amounts are likely to be too small to induce any kind of positive effect	[38]
Fluoride in drinking water	The anti-fluoride community has been extremely vocal in the "toxic" effects of fluoride metabolites in drinking water, even though the addition of this element to drinking water has been one of the primary reasons for improved dental health and related cardiovascular diseases	[39]
Hypermobile EDS	A joint hypermobility condition with no genetic etiology. This may not be a discrete syndrome but a point on the spectrum of hypoelasticity and hyperelasticity of joints	[40]
POTS	An increase in heart rate when moving from a supine to a standing position, with associated clinical symptoms. Unfortunately, other non-related symptoms or signs appear to be frequently lumped in with this diagnosis	[40]
MCAS	Overactive mast cells. This condition is being blamed for a large number and vague and unclear symptoms of significant variability, even though there is no pathophysiologic mechanism by which mast cells may actually participate in real life	[40]
Relationship between hEDS, POTS, and MCAS	This is gaining momentum to explain incorrectly why people have symptoms of fatigue, loss of concentration, vague abdominal complaints, etc. There is no scientific evidence that such a relationship exists	[40]
Systemic candidiasis	This condition has been claimed by many people in search of a reason for their ailments who ignore the complete lack of scientific evidence. In fact, systemic candidiasis only affects immunocompromised individuals	
Leaky gut syndrome	Another condition which has never been proven to exist but is latched onto by patients who have abdominal symptoms. In fact, adding such a label to their medical history appears to give these patients some satisfaction, without offering any worthwhile treatment	
Gluten sensitivity	Without any scientific evidence, a culture of non-celiac gluten sensitivity has permeated twenty-first century life. Ingestion of gluten has been associated with a myriad of symptoms, from headache to abdominal pain, to diarrhea and even fatigue or weakness. People will believe what they want, and unfortunately, untruths spread wildly and soon gain mainstream acceptance in the absence of any scientific corroboration. Although not a perfect test itself, a challenge to gluten may in some cases rule in or rule out an intolerance to gluten, but because many of the symptoms these patients experience are vague and unquantifiable, not to mention very variable from a timing standpoint, challenge may not always be helpful	[41, 42]

Unproven Tests of which Medical Practitioners and the Public Should Be Wary

As mentioned above, testing for urinary mycotoxins is gaining popularity, but this has no clinical value. Testing one's home is expensive and it has been found that visible mold does not correlate with results in air and dust sampling. [26, 27] In another study, asthma symptoms did not correlate with skin sensitivity to molds or allergen concentration in the air. [28]

People develop IgGs to foreign proteins when they are exposed to them. IgGs do not measure allergies, and thus should not be used in the diagnosis of IgE-mediated mold allergy. Recently, some labs have been advertising IgG testing to *Candida*, and are offering in-home IgG testing to foods and *Candida*. There is no scientific basis for this type of testing. Testing for antibodies to mycotoxins have not been validated and has no clinical utility. [29] Testing for specific IgG4 to molds also does not provide any useful clinical information. There is a high cost to the patient and no clinical benefit to performing any of the above tests.

There have been reports that IgG precipitins may help diagnose fungal hypersensitivity pneumonitis, such as ABPA, but even this is unsubstantiated. [30] In fact, IgG antibody testing and IgG precipitins to *Aspergillus* are tests that have not been standardized, and interpretation of the results may be challenging. [31]

Conclusions

There is no evidence for the relationship between toxic mold and any illness in humans. The continued belief in this myth is perpetuated by those charlatans who believe that measles vaccines cause autism, that homeopathy works, that fluoride in the water should be removed, that chronic regional pain syndrome is a real disease, or that there is a relationship between mast cell activation syndrome, hypermobile Ehlers-Danlos syndrome, and postural orthostatic tachycardia syndrome. These are unproven conditions or relationships without scientific evidence (Table 3). The result of these popular diagnoses or treatments is that science is often left by the wayside and the truth becomes a matter of opinion. Mycotoxicosis is one of these situations in which there is no scientific evidence for its existence.

It is easy to understand why patients who have been suffering with vague symptoms for years and who have been searching for a reason will latch onto a false disease in the hopes that this will provide an explanation for their symptoms. It does not matter to them that there is no basis for their belief. It is often easier for a physician to go along with this as well, rather than confront the patient about the real problem, whether that be physiologic or psychological in nature. This may help the physician gain the patients trust initially, but in the long run, will probably do more harm than good. It is time to stop talking about conditions and treatments that are not based on science and have no plausible pathophysiologic mechanism.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval There is no funding so no ethical approval is required.

Informed Consent No informed consent is required.

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