Global Indoor Health Network (GIHN)

"Working Together for Healthy Indoor Environments"

PO Box 777308
Henderson, NV 89077-7308

globalindoorhealthnetwork.com

Diagnosis and Treatment of Illness Caused by Contaminants in Water-Damaged Buildings

Table of Contents

Overview

Astute physicians and healers have been aware of the existence of environmental toxins for over a thousand years. The list of substances, both naturally occurring and manmade, which may cause harm to the human organism, is continually growing. For details on many different types of environmental toxins, see our paper on "Indoor Air Contaminants." Our papers are posted on our website.

Curiously, while heart disease, cancers and rare exotic illnesses frequently grab headlines, illness due to environmental sources, though incredibly common, often receive little or no media coverage.

Typically, little education is offered to allopathic physicians in their medical training on this subject. Hence, there is poor understanding of the concept that our environment is capable of slowly poisoning its inhabitants. However, there are thousands of research papers on this topic that describe the significant health effects of indoor air contaminants.

The significant risk of illness caused by contaminants inside water-damaged homes, schools and businesses is summed up in the following statements from the World Health Organization:

> Indoor air pollution – such as from dampness and mould, chemicals and other biological agents – is a major cause of morbidity and mortality worldwide.[1]

> Indoor dampness is estimated to affect 10–50% of indoor environments in Europe, North America, Australia, India and Japan. In certain settings, such as river valleys and coastal areas, the conditions of dampness are substantially more severe than the national averages for such conditions.[1]

Indoor air pollution – such as from dampness and mould, chemicals and other biological agents – is a major cause of morbidity and mortality worldwide.

As we will discuss throughout this paper, the contaminants in water-damaged buildings can include molds, mycotoxins, bacteria, volatile organic compounds (VOCs) and other types of contaminants. Each of these contaminants can cause serious health effects.

Because these contaminants can cause health effects through absorption or inhalation, there is the potential for many different body systems to be affected. Research papers on this topic describe a wide range of symptoms. In a research paper by Dr. Harriet Ammann, she lists the potential system effects in the following categories: vascular, digestive, respiratory, nervous, cutaneous, urinary, reproductive and immune.[2-3] In regard to mycotoxins, Dr. Amman states:

> Mycotoxins are nearly all cytotoxic, disrupting various cellular structures such as membranes and interfering with vital cellular processes such as protein, RNA and DNA synthesis.[2-3]

In a report by the U.S. Environmental Protection Agency (EPA), they describe the health effects as follows:

> Health effects from indoor air pollution cover the range of acute and chronic effects, and include eye, nose, and throat irritation, respiratory effects, neurotoxicity, kidney and liver effects, heart functions, allergic and infectious diseases, developmental effects, mutagenicity, and carcinogenicity.

The extent of this problem is tremendous, with millions of homes, schools and buildings around the world affected by water damage. Here are a few key statistics:

- 10-50% of all indoor environments in Europe, North America, Australia, India and Japan are affected by indoor dampness (World Health Organization--WHO); the report says the numbers are much higher in some areas
- 50% of U.S. schools have indoor air quality problems (U.S. Environmental Protection Agency--EPA)
- 30-50% of all U.S. homes (U.S. Consumer Product Safety Commission--CPSC)
- 47% of U.S. homes (Research study supported by the U.S. EPA)
- 45% of U.S. office buildings (U.S. EPA)
- 40% of U.S. homes have health and safety hazards (National Center for Healthy Housing (NCCH). This statistic is based on a report from the U.S. Department of Housing and Urban Development (HUD).

  The study reveals that 35 million – 40% – of metropolitan homes in the U.S. have one or more health and safety hazards. The study found that the most common housing problems identified include water leaks, roofing problems, damaged interior walls, and signs of mice.
- 33-50% of all structures (U.S. EPA)
- 30% of buildings have indoor air quality problems (U.S. Occupational Safety and Health Administration--OSHA)
- 20-26% of nursing homes, hospitals and outpatient departments (a report from Finland); percentages also provided for houses, apartment buildings, schools and offices

It is staggering to comprehend the enormous impact on our global society due to medical costs, lost productivity, repairs needed. To see the references for these statistics and to learn more about the statistical and economic impact of this issue, read our paper titled "Global Burden of Indoor Air Contaminants." Our papers are posted on our website.

When you look at the immense scope of the problem, you can understand why we are presenting a comprehensive view of this topic. Please help us to share this important information on diagnosis and treatment with government agencies, medical organizations, physicians, patients and others around the world.

In this paper, we will provide an extensive list of references to published research papers. However, this list is certainly not all inclusive. There are thousands of research papers available.

The Name for this Illness

Mold illness, mold-related illness and biotoxin-related illness are euphemisms for the same disease. Some of the names for this illness in the U.S. are Mycotoxicosis, Mixed Mold Mycotoxicosis, Indoor Mold Sensitivity and Toxicity, Toxicant Induced Loss of Tolerance (TILT) or Chronic Inflammatory Response Syndrome due to Water Damaged Buildings (CIRS-WDB or CIRS). In Finland, it is called Dampness and Mold Hypersensitivity Syndrome (DMHS).[4]

In our 2012 position statement, rather than favor one group's name over another's, we presented a temporary name for the illness -- Multi-system Exposure Related Illness (MERI, pronounced "meer-ee") -- to refer to the disease as it points to the multi-systemic nature and indoor environmental triggers which include, but are not limited to, toxins, microbial secondary metabolic products, particulates and the microbes themselves.[5] This illness also recognizes that toxins other than mold or microbial secondary metabolic products may create comparable symptomatology, presumably through the same or similar pathways.

Two new names for this illness were introduced in 2016 and 2017.

In early 2016, Dr. Michael Gray presented a new name for illness caused by exposure to toxins and contaminants in our environment. He refers to this illness as Cumulative Organic Chemical Hyper-Toxicity (COCHT). He explains how toxins can accumulate in our bodies and how genetic deficiencies make it difficult for some people to neutralize, metabolize or eliminate these toxins. Dr. Gray is with the Progressive Healthcare Group[6] in Benson, Arizona, and more information about COCHT can be found in his educational videos.[7]

In September 2017, a new nonprofit organization was created by a group of doctors working in this field of medicine. The name of this nonprofit is International Society for Environmentally Acquired Illness (ISEAI). As the name implies, they are referring to this illness as Environmentally Acquired Illness (EAI).[8] From the ISEAI website:

> It is no secret that many patients today are sicker and their illnesses are more complex than they were even 20 years ago. Even so, doctors are expected to diagnose and treat patients in less time and with more restrictions. Medical appointments are so brief that patients can present only one or two complaints at a visit. Few doctors have the time or ability to look at a patient's whole health picture. Our chronically ill patients are sick, exhausted, in pain, anxious, not sleeping, digesting or thinking properly. They have been misdiagnosed or told their problems were "in their heads." ISEAI aims to help the medical community to better serve these patients.[8]

For the purpose of this paper, we will not be using a specific name for the illness. We prefer to leave it open until the medical community comes together to select a name that can be accepted and adopted by all involved.

It is important to acknowledge the large number of individuals and families around the world who are being affected by illness caused by exposure to these indoor contaminants in water-damaged buildings. A quick search on the Internet will provide results of countless personal stories from people being harmed by the contaminants in water-damaged buildings (with new stories occurring every day). You can read a small selection of their stories on our website under the heading "Personal Stories."

This illness is not currently being tracked by the medical community, so an accurate count of patients is not available. However, given the multi-system, multi-symptom nature of this illness and the extensive list of health effects, there are likely millions of individuals with this illness throughout the world.

Most physicians will not recognize the illness because they are uninformed about the variable multi-system presentations or have been misinformed about the serious health risks of molds, mycotoxins and other contaminants inside water-damaged buildings.[9-42]

This illness is currently known by several names including Mycotoxicosis, Mixed Mold Mycotoxicosis, Indoor Mold Sensitivity and Toxicity, Toxicant Induced Loss of Tolerance (TILT), Chronic Inflammatory Response Syndrome due to Water Damaged Buildings (CIRS-WDB or CIRS), Cumulative Organic Chemical Hyper-Toxicity (COCHT) or Environmentally Acquired Illness (EAI). In Finland, it is called Dampness and Mold Hypersensitivity Syndrome (DMHS).

Typically, one or two systemic problems predominate while several other systems are regularly involved, as evidenced by simultaneous abnormalities among multiple targeted diagnostic tests evaluating multiple organ systems. Patients, who complain of intense fatigue on a daily to almost daily basis, are frequently diagnosed with Chronic Fatigue Immune Dysfunction Syndrome (CFIDS), or Myalgic Encephalomyelitis (ME), with the diagnosing clinicians often claiming wrongfully that their patient is suffering from a somatoform or "functional" illness. These physicians have no appreciation that its real cause rests with mitochondrial dysfunctions caused by the direct poisoning of cellular respiration by mycotoxins which interfere with oxidative phosphorylation and cause deficiency states involving multiple enzymes of the electron transport chain pathway, or aerobic metabolism, also known as cellular respiration.

It is a tragic, time-honored habit of many physicians ignorant of the underlying physiologic defects causing the pathology of their patients, to claim that—since the explanation for the problem is not in their heads—the problem must be in their patients' heads. They psychologize their patients' problems and then impose inappropriate and often damaging treatments on their patients. These treatments are often court mandated and use mind-numbing and often neurotoxic psychotropic, anti-depressant, anti-psychotic or anxiolytic pharmacologic agents---the scientific basis for which is often absent or marginal, at best.

Those with a chief complaint of severe and chronic muscular pains are wrongly diagnosed as having fibromyalgia, without realizing that this condition which--according to the U.S. Centers for Disease Control (CDC)--occurs in 3.5 to 4.5% of the general population and 10.9% of the chemically challenged (Multiple Chemically Sensitive—MCS) patients. And, in

20% of patients whose illness arose out of exposures occurring in water-damaged, microbial-amplified, bioaerosol-laden indoor environments, confirming a causal relationship with those environmental factors, at a level of reasonable medical and scientific certainty.

Patients with recurrent abdominal pains, with or without diarrhea, are labeled with irritable bowel syndrome (IBS), Crohn's Disease, or Inflammatory Bowel Disorders, of unknown cause, without regard to the growing body of data confirming involvement of fungal colonization of the gut lining.

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When the primary concern relates to odd neurologic symptoms, the patient may be misdiagnosed as having multiple sclerosis (MS), Parkinson's disease, Autism Spectrum Disorders, Attention Deficit Disorder (ADD), Attention Deficit Hyperactivity Disorder (ADHD), Choreoform Movement Disorders, Seizures, Amyotrophic Lateral Sclerosis (ALS), and other Degenerative Neurologic Disorders "of unknown origin." However, all of these patients show multi-systemic symptomatology frequently arising from exposure occurring in the contaminated indoor environments of our homes, schools and workplaces.

Pidgeon-holing the sufferer into a single-system diagnosis requires ignoring or minimizing many other symptoms and systemic clues. Patients are frequently told they are depressed, anxious and need psychiatric medications, while the central environmental history of home and work was never explored. Others are told they are somaticizing (or worse – malingering), that they need to "learn to live with it," or that it's "all in their head."[40,42]

Another tactic used by the naysayers to deny this illness is to blame it on psycho-social factors. On rare occasions, the patient will be told honestly by the practitioner that (s)he doesn't know what is wrong.

For additional information about the government agencies, insurance companies, medical organizations, defense experts, defense attorneys and others who spread their false claims and refuse to admit the truth about this illness, read our paper titled "Discussion of Naysayers and Deniers." Our papers are posted on our website.

The following statements in a 2017 research paper in Finland provide a good summary of this situation and how it is harming patients and keeping them from getting proper medical care:

> Mold-related illness should not be viewed as a so-called medically unexplained syndrome, as has been claimed. In our opinion, providing these patients with cognitive or behavioral therapy is medically unethical—it represents a denial that mold-exposed

individuals are suffering from a somatic illness. Moreover, cognitive/behavioral therapy is not effective.

We can assume that providing the mold-exposed patient with only psychotherapy in combination with high dosages of corticosteroids while he/she continues to live or work in a hazardous environment is inappropriate "medication;" in fact, it will aggravate their risks of suffering severe morbidity and even dying.

Providing the mold-exposed patient with only psychotherapy in combination with high dosages of corticosteroids while he/she continues to live or work in a hazardous environment is inappropriate "medication;" in fact, it will aggravate their risks of suffering severe morbidity and even dying.

On the basis of the present data, we think that it is irresponsible to claim that indoor molds cause only transient irritation symptoms and pose only a 1.5-fold risk for the development of asthma. Even though more and more knowledge is available on the mechanisms underpinning the health hazards associated with moldy environments, mold-related disease is still called a "non-disease," or "somatoform disorder," with some physicians trying to label it as a "fashionable" disorder, or stating that its sufferers are exhibiting hysteria.

Mold-related illness is a somatic disorder; the symptoms are physical, not psychosocial problems, although this has been claimed for almost 20 years. In most cases, later it can become a psychosocial problem as patients suffer mental distress from their failure to convince physicians that they are ill.

Our data show that occupying a contaminated building for even 2–3 years (either a home or a school) can seriously impair the well-being of potentially healthy individuals, even to the extent of loss of life. Therefore, any attempt by governmental/medical authorities to deny the serious effects of toxic molds on human health should be combatted.[40]

Although there is not yet one common name for this illness, it is our opinion that a single unifying name would benefit all the various vested communities (treating physicians, researchers and sufferers).

Eventually, the medical community will come together and agree upon a name that will be easily remembered and resonate with lay people, media and scientific personnel.

We recently saw this occur when a new name was proposed for an illness known as Myalgic Encephalomyelitis (ME) or Chronic Fatigue Syndrome (CFS). In February 2015, the Institute of Medicine (IOM) offered a new name for ME/CFS---Systemic Exertion Intolerance Disease (SEID).[43] This 2015 report from the IOM included the following recommendations:

The primary message of this report is that ME/CFS is a serious, chronic, complex, and systemic disease that frequently and dramatically limits the activities of affected patients.

In its most severe form, this disease can consume the lives of those whom it afflicts. It is "real."[43]

We believe a new name and similar message will eventually be announced in regard to illness caused by exposure to contaminants in water-damaged buildings. A single, unifying name would give sufferers of this illness precious hope by accurately identifying the composite causes of their symptoms and enabling them to receive appropriate medical care. This would also set the stage for new, accurate messages for health professionals and government agencies around the world, and the naysayers' false claims would disappear and be replaced with the truth.

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In summary, this illness is a multi-symptom, multi-system disease occurring in many people due usually to long-term exposure to the interior of water-damaged buildings (WDB). While there are differing opinions on the best diagnostic and therapeutic approaches, it is clear from the literature and from practice that this disease exists and significant relief can be obtained by most sufferers with avoidance of further exposure and appropriate treatment.

The tide is turning. Knowledge and awareness of this illness is spreading, and the diagnosis and treatment protocols are being shared around the world.

Water-Damaged Buildings

Mold and bacteria are ubiquitous, inside and outside of buildings. Adding water provides the missing ingredient needed for the explosive microbial growth, known as amplification, found in water-damaged buildings (WDB).

Water damage inside buildings is mainly caused by natural disasters or human activities (e.g., lack of repair and maintenance in schools and office buildings). The determining factor for fungal growth on building materials is water activity. This is why eliminating the source of water intrusion is the first step in remediation of water-damaged buildings.

> Dampness problems in homes, office buildings, schools and other nonindustrial buildings may develop moisture and dampness problems which can lead to the growth of mold, fungi and bacteria, the release of volatile organic compounds, and the breakdown of building materials.[44]

Construction materials in the form of sheetrock, drywall, wood, etc. offer a great amount of food resources for indoor molds and bacteria.[45-51]

> Molds can obtain nutrients and moisture sufficient for growth from water-affected building materials such as wallboard and insulation materials, as well as carpets, furniture, and clothing. They feed on dead organic matter and, provided with sufficient moisture, can live off of many materials found in homes, such as wood, cellulose in the paper backing on drywall, insulation, wallpaper, glues used to bond carpet to its backing, and everyday dust and dirt.[50]

Buildings become water-damaged when water intrudes via numerous pathways including leaking roofs, inadequate vapor barriers, indoor plumbing leaks, faulty HVAC (heating, ventilation and air conditioning) systems, condensation drainage, and intrusions into basements and crawl spaces through several mechanisms. Another common source of water damage is caused by delayed or insufficient maintenance of homes, schools or office buildings or improper design of HVAC systems (e.g., insufficient cooling capacity).

Mold often occurs in schools during the summer because the school districts decide to turn off the air conditioning which causes the humidity to rise, or they neglect to keep up with building repairs and maintenance.

Another important consideration regarding water damage is the relative humidity level inside buildings. Most guidelines recommend that the humidity levels indoors be kept below 50-60%. The U.S. EPA says that indoor humidity should ideally be kept between 30-50%.[51]

> Relative humidity also affects spore release for some molds (e.g., *Aspergillus* and *Penicillium*), with spore release occurring with lowering humidity after initial growth at high humidity levels. One reviewer concluded that "the worst-case scenario for the development of an indoor mold problem involves a series of water intrusion events that allow large quantities of biomass and mycotoxins to form, then a period of drying that promotes the dispersion of spores and colony fragments, followed by their deposition throughout the building."[50]

As mentioned earlier in this paper, the scope of this problem is tremendous. There is some guidance available on the ways to build mold-resistant homes and there are others working on creating mold-resistant building materials, but much more progress could be made in this area when the government agencies, insurance companies, medical organizations and naysayers acknowledge the health effects of contaminants in water-damaged buildings.

Pathophysiology

Illness due to exposure in WDB buildings results from a combination of factors and includes the direct effects of toxins, chronic inflammation and colonization and infection of microbial agents. In an amplified system, there is unchecked expansion of numerous species of

molds, bacteria, *Actinomycetes* and *Mycobacteria*, and unfettered production of spores and secondary metabolites such as endotoxins, β-D-glucans, spirocyclic drimanes, trichothecenes, aflatoxin, ochratoxin, satratoxins, galactomannans, hemolysins, fine particulates, etc., as well as Volatile Organic Compounds (VOCs) from the building materials and microbial VOCs (MVOCs)which are released from damp cavities, through sheetrock, into the air the inhabitants breathe.[52-68]

> Persons can be exposed to mold through skin contact, inhalation, or ingestion. Inhalation is usually presumed to be the most important mechanism of exposure to viable (live) or nonviable (dead) fungi, fungal fragments or components, and other dampness-related microbial agents in indoor environments. The majority of fungal spores have aerodynamic diameters of 2--10 µm, which are in the size range that allow particles to be deposited in the upper and lower respiratory tract. Inhalation exposure to a fungal spore requires that the spore be initially aerosolized at the site of growth. Aerosolization can happen in many ways, ranging from disturbance of contaminated materials by human activity to dispersal of fungi from contaminated surfaces in heating, ventilating, and air-conditioning (HVAC) systems.[69]

Although the naysayers claim that illness occurs only due to ingestion (i.e., eating foods with mycotoxins), inhalation also causes illness and is a greater risk because the toxins enter the blood-brain barrier and can affect multiple organs.[1-42,69]

Inhalation exposure is a greater risk (than ingestion) because the toxins enter the blood stream and can affect multiple organs and can bypass the blood-brain barrier.

> Inhalation exposure gives direct access to the general circulation through the alveoli, without a first pass through the liver for detoxification as the ingestion route does. Inhalation exposure also provides a pathway to the central nervous system along the olfactory and trigeminal nerve axons in the nasal sensory epithelium that bypasses the blood–brain barrier. Deposition of these small particles occurs throughout the respiratory tract, but especially in the alveoli where transport to the bloodstream largely occurs, resulting in toxin distribution to other systemic target organs.[14]

The literature provides ample evidence that exposure to the interior of WDB leads to increases in upper respiratory syndromes, allergies and increased incidence of asthma with further triggering of asthma flares.[70-107]

There is also significant evidence regarding the neurological effects, even though the government agencies and naysayers continue to deny it.[108-109] Direct neurotoxicity and immunotoxicity of some mycotoxins have also been clearly demonstrated in the literature.[2-15,17-23,30-40,110-143] We will discuss this topic further throughout this paper.

In addition to the respiratory and neurological effects, dermal and ocular effects can result from exposure to water-damaged environments and can contribute to illness.[2-3,144-153]

Many occupants of water-damaged buildings and remediation workers experience dermal effects including skin rashes and lesions. A 2017 study found that:

> The T-2 mycotoxin is distinctive in that systemic toxicity can result from any route of exposure, i.e., dermal, oral, or respiratory. As a dermal irritant and blistering agent, it is alleged to be 400 times more intoxicating than sulfur mustard.[148]

Another study by Chattopadhyay, et al, also discussed the dermal effects, as follows:

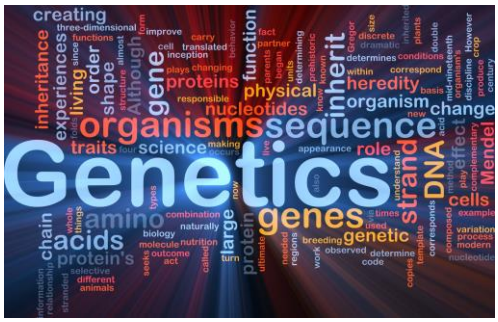
> Dermal exposure to T-2 toxin caused severe cardiotoxicity in experimental Wistar rats. Electrocardiography studies showed the conduction abnormalities including prolongation of the QT and corrected QT interval, shortening of the PR interval, and tachycardia. Biochemical studies also reported the toxicity of T-2 toxin. T-2 toxin induced acute cardiotoxicity in rats and characterized by significant ($p < 0.05$) elevation of serum troponin I, creatine kinase (CK) isoenzyme MB, CK isoenzyme NAC, and lactate dehydrogenase as compared to control rats.[150]

Ocular effects have also been discussed.[143,145-146,151-152] Muzi, et al, discussed the effect on tear stability, as follows:

> Employees in the sick building complained more frequently of ocular symptoms, upper-airway disturbances, and general and respiratory symptoms than did employees in the comparison buildings. Stability of tear film was significantly reduced ($P< 0.01$) in the employees in the sick building compared with employees in the comparison buildings.[151]

Tear-film stability, the ability of the tears to adhere and spread easily and evenly across the ocular surface and remain intact, without evaporation causing dry spots in between blinks. The tear film protects and lubricates the ocular surface, as well as providing other functions such as providing nutrients, aiding with immunity and refraction, to name a few. Tear-film stability is important for ocular health.[153]

Individual genetic factors as well as underlying health and nutritional status also contribute to an ongoing inflammatory response and symptoms even after exposure to the water-damaged environment has ended.



Risk is affected by genetic factors - including variations in cytochrome P450 detox pathways, glutathione pathways and HLA (human leukocyte antigen) genotype which may affect an individual's ability to detoxify - as well as response to presenting antigens.[17-18,33-35,154-170]

> Evidence for the role of oxidative stress in the pathophysiology of mycotoxin-related illness is increasing. The glutathione antioxidant and detoxification systems play a major

role in the antioxidant function of cells. Exposure to mycotoxins in humans requires the production of glutathione on an "as needed" basis. Research suggests that mycotoxins can decrease the formation of glutathione due to decreased gene expression of the enzymes needed to form glutathione. Mycotoxin-related compromise of glutathione production can result in an excess of oxidative stress that leads to tissue damage and systemic illness.[161]

The role of glutathione in detoxification is explained further by Sadiq, et al, as follows:

Glutathione plays critical role in xenobiotics detoxification. It also plays a role in conjugation of toxic metabolites generated in the cell during phase I reactions. It participates in glyoxylase system, protein regulation and expression of gene through disulfide exchange reactions, metabolism of estrogens, prostaglandins and leukotrienes, maturation of iron-sulfur clusters of diverse proteins and operation of certain transcription factors. Glutathione plays indispensable role in cytoprotection, detoxification, protein protection and redox-signalling.[169]

While a massive acute exposure can lead to this illness, the most common mechanism is chronic exposure to low level toxins leading to an inflammatory response in the body. Inflammation is not caused by the typical path seen with infecting agents. The inflammatory mechanism in this illness is unlike the typical infectious agent that presents antigens to dendritic cells, and antibodies result. Rather, HLA-DR (DR portion of the HLA genome) does not facilitate antigen presenting cell (APC) recognition of antigens as foreign.

While a massive acute exposure can lead to this illness, the most common mechanism is chronic exposure to low level toxin leading to an inflammatory response in the body.

In this model, the toxin(s) bind to Toll-like (adipose cells) and non-Toll receptors acting as pattern-recognition receptors then activate the innate immune system in the form of the mannose binding lectin (MBL) pathway[170-176] of the complement system through a secondary messenger scheme.

This leads to continuous stimulation of the MBL pathway without an effective "turn-off switch" (since no foreign particle was presented to APCs to be cleared). As such, the MBL runs smoothly and efficiently around the clock for weeks, months, years and even decades, all the while producing pro-inflammatory cytokines[17-18,34,177-181] with the ultimate intent of destroying "something."

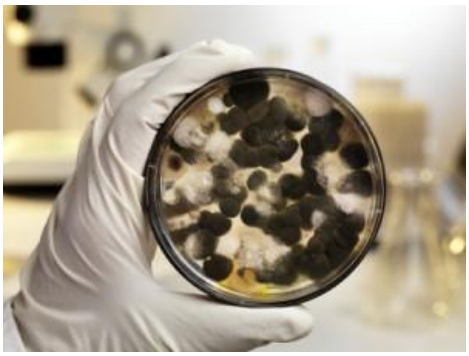
Since there is no foreign target being presented or opsonized for destruction, eventually those cytokines will cause damage to the host. Innate immune abnormalities are often demonstrated in patients by elevated TGF-β1 (transforming growth factor, beta 1), C4a (the activation product of the complement protein C4) and/or MMP-9 (matrix metallopeptidase 9) levels.[17-18,30,35,177-182]

Currently, our detection and testing methods are not sensitive enough to determine which individual toxin, or group of toxins, causes illness for each individual patient. Different patients likely will have individualized susceptibilities and each water-damaged building has its own unique set of pathogenic, toxin-producing microbes and micro-particulates.

What is clear from re-exposure studies, however, is that certain buildings will cause a rapid reproduction of symptoms and abnormal lab studies in patients when re-exposed off therapy.

Of note, it does not matter which toxin is offending. In the primordial milieus found in WDB, many toxins, particles, fragments, spores, etc. are being released, and each building will be different.[52-53,57,183-187] The number of cell wall or desiccated colony fragments released into the air will be many hundreds of times greater than spores.[52-53]

Particulates shed from molds include spores, fragments of mycelia and nano-particulates. Field studies of water-damaged homes have shown concentrations of nano-particulates in indoor dust that are at least 1000 times or greater than the indoor air mold spore counts.[53]



Different and multiple species of molds, *Mycobacteria*, *Actinomycetes* and other bacteria will be found—many releasing different secondary metabolites including mycotoxins and endotoxins.[15,50,52-53,57,183-187]

The bio-contamination resulting from water intrusion includes: (1) molds; (2) bacteria; (3) microbial particulates; (4) mycotoxins; (5) volatile organic compounds (non-microbial [MVOCs] and microbial [VOCs]); (6) proteins (e.g. secreted enzymes, haemolysins and siderophores); (7) galactomannans (extracellular polysaccharides or EPS); (8) 1-3-D-b-glucans (glucans) and (9) endotoxins (lipopolysaccharides [LPS]).

Furthermore, when the synergism and interactions of the biocontaminants are considered, it can only be concluded that multiple systemic health effects in humans and animals are not only occurring but are scientifically and medically explicable.[52]

Unlike animal cells, which digest nutrients in their interior, fungi -- including yeasts and molds -- actively secrete their digestive enzymes onto their surface in a process called exodigestion. Exodigestive enzymes are proteins that coat the surface of desiccated fungal particulates making them extreme antigens, or immune system stimulants. VOCs and microbial VOCs (MVOCs) may also be released.

It is often difficult (and not essential) to discern which toxin or combination of toxins cause harm. That there are one or more toxins causing harm to the exposed human host is the crucial matter.

Put another way, Dr. John Snow (the "Father of Modern Epidemiology") noted in 1854 that a cholera outbreak centered around the common use of the Broad Street public well. He removed the pump's handle and the outbreak dissipated. *Vibrio cholerae*, the organism responsible for cholera, was coincidentally discovered the same year but was not widely known for another 30 years.

Would it have been wise for Dr. Snow to withhold his actions for 30 years until the precise bacterial agent could be identified?

Likewise, Ignaz Semmelweis postulated the theory of hand washing between medical procedures in 1847. He published several works on the subject and was widely criticized. However, Louis Pasteur and his microscope proved the existence of bacteria and refuted the idea of spontaneous generation in the 1860s.

Several years later, in 1867, Sir Joseph Lister published on the use of "antiseptic principles" (like hand washing). After 28 years, antiseptic practice finally became the standard of care.

Should the medical community wait 28 years or longer to develop the technology to determine which individual toxin(s) is (are) causing this illness, or should buildings be fixed and patients be treated now? The question is rhetorical; the answer is obvious.

Should the medical community wait 28 years or longer to develop the technology to determine which individual toxin(s) is (are) causing this illness, or should buildings be fixed and patients be treated now? The answer is obvious.

In the CIRS-WDB model, toxins from the interiors of WDB are inhaled, transported to the blood and lymph-bile systems, and ultimately find their way to adipose cells to trigger the mannose binding lectin pathway of the complement system. More and more pro-inflammatory cytokines are released into the bloodstream.

Initially, neuroregulatory and immune peptides such as Vasoactive Intestinal Polypeptide (VIP) and Melanocyte Stimulating Hormone (MSH) exert control on the immune system to mitigate the damage. However, in an epic tug of war, constant cytokine production eventually outruns overproduction of VIP, MSH and others (ADH or Anti-diuretic hormone included).[17-18,123,33,35,158,192-195] When the production of these hormones is overwhelmed, it is possible that low levels ensue similar to depleted insulin production in a patient becoming a Type II diabetic.

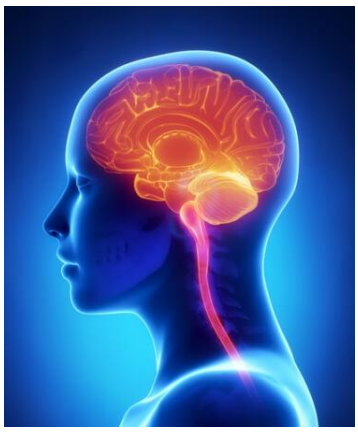
Pro-inflammatory cytokine production becomes relatively unopposed leading to multisystem damage likely mitigated by T_h17 lymphocytes (T helper 17). Chronic systemic inflammation leads to localized degradation of the blood brain barrier (BBB) allowing T_h17 cells access to the cerebral blood supply and the parenchyma itself. TGF-β1 (an anti-inflammatory cytokine) appears to play an important role.

Normal CD4+ (cluster of differentiation 4) cells in the presence of normal or elevated TGF-β1 promote commitment to CD4+CD25+ (T_h3 regulatory or T_{REG}) lymphocytes which decrease auto-immunity. However, low levels of TGF-β1 in the presence of pro-inflammatory interleukins (IL-6, IL-21 or IL-23) push naïve CD4+ cells' commitment to T_h17 cells,[196-204] known to promote auto-immunity and numerous well-described autoimmune diseases.

Summing up, the WHO 2009 report states (p. 85):

> Many of the health effects may result from recurrent activation of immune defence, leading to exaggerated immune responses and prolonged production of inflammatory mediators. Overproduction of these compounds damages the surrounding tissues and may manifest itself as chronic inflammation and inflammation-related diseases, such as asthma (Martin, Frevert, 2005).[1]

In addition, there are degenerative neurologic changes because some mycotoxins are directly neurotoxic causing global neurologic injury manifesting as visual contrast deficits, balance problems, cognitive deficits, abnormal pain patterns, recurrent numbness and tingling, extraordinary skin sensitivity (even on the order of that seen in complex regional pain syndrome), etc.



Cognitive deficits and behavioral issues have been shown in persons with systemic inflammation as well as in those exposed to mold and mycotoxins in water-damaged buildings.[17-18,29,34,109-110,115,138-143,177,206-214]

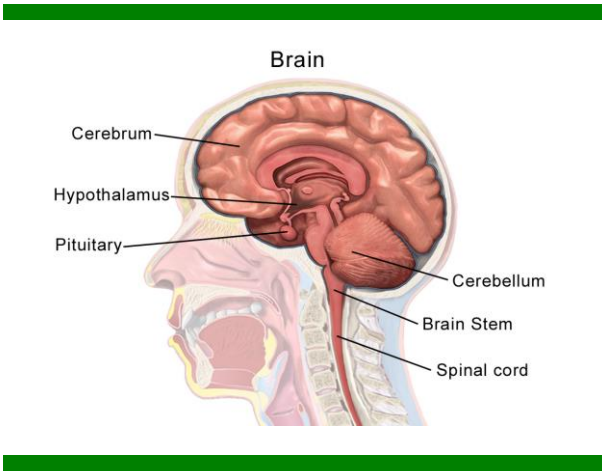
IgG and IgM (immunoglobulin G and M) antibodies to several neuronal peptides have also been demonstrated in persons with documented mycotoxin exposure and neurologic dysfunction.[29,31,37,111,117,133,211,215]

In these patients, Magnetic Resonance (MR) Spectroscopy shows reproducible deficits consistent with brain hypoperfusion[158,212,216] which reverse after therapy. Some of these patients, without a positive myelin basic protein from cerebrospinal fluid, may be incorrectly diagnosed as having multiple sclerosis.[113,217]

Treating physicians have observed there is an over-representation of patients with midbrain movement disorders—choreas, obsessive compulsive disorder and Tourette's syndrome—among these patients.[107,111,114-115,119,141,212-220] Basal ganglia lesions found on CT

(computerized tomography) were predictive of movement disorders in people from 13 Chinese provinces who ingested sugar cane contaminated with *Arthrinium s.*[221] Additional mid-brain lesions are also associated with the autonomic nervous system.[222-228]

This illness often reflects endocrine disruption. It is important to understand that the hypothalamus and pituitary are very much affected. The hypothalamus, as seat of control over the autonomic nervous system, many of the body's "set points" (such as temperature control) and the endocrine system via the pituitary, is the structure of most ultimate importance in homeostasis of nearly all body functions.



As such, almost every system of the body can be affected by this illness and the multiple and varied presentations stem from the fact that different patients, while having many symptoms crossing many systems, tend to have 1 or 2 predominating systemic difficulties. As stated above, if a practitioner focuses attentions primarily on those 1 or 2 systems, (s)he is likely to neglect the larger picture which involves many systems.

Looking again at the CIRS-WDB model regarding the hypothalamus and pituitary, abnormalities are frequently detected which are believed to be the result of decreased production of MSH. Stimulation of leptin receptors activates the pro-opiomelanocortin (POMC) pathway. Weight gain is frequently seen in those exposed to water-damaged buildings. This could be in part due to leptin resistance which often develops as a result of pro-inflammatory cytokine action, pathway overusage due to increased need of MSH, and/or as a response to increased adiposity in those already overweight. Increasing leptin resistance with subsequent decreased MSH alters the point of satiation. Patients often gain significant weight which is not responsive to diet and exercise.[17-18,23,33,35,158-160,228-236]

MSH modulates mucous membrane immune responses which work against nasal carriage of biofilm forming multiple antibiotic resistant forms of coagulase negative staph (MARCoNS). In turn, MARCoNS produce hemolysin which cleaves MSH. β-endorphins, adrenocorticotropic hormone (ACTH) and MSH are all produced in the POMC pathway. Reduction of this pathway's use means less of the body's most potent pain reliever is made.

Many patients suffer chronic pain. MSH may also be critical for restorative sleep. Some patients can sleep 10-12 hours but not feel rested upon waking. Many have much more trouble falling asleep and with early awakening.

> Most likely, mycotoxins exert some rigorous effects on the circadian rhythmic processes resulting in sleep deprivation to which an acute and transient increase in NKC activity is observed. Depression, psychological stress, tissue injuries, malignancies, carcinogenesis,

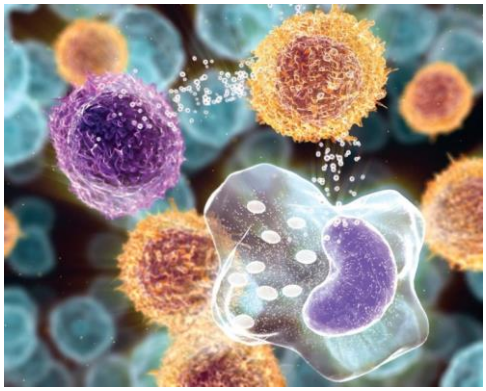
chronic fatigue syndrome, and experimental allergic encephalomyelitis could be induced at very low physiological concentrations by mycotoxin-induced NKC activity.[112]

Functional hormonal dysregulation is identified in these patients. Abnormal laboratory testing frequently includes depleted ACTH, MSH, ADH and TSH. ADH depletion leads to chronic dehydration and elevated levels of histamine (with resulting symptoms), which, in turn, contributes to depleted hypothalamic production of TRH. Thus, TSH is normal instead of elevated, and functional hypothyroidism is often a missed diagnosis. Or, still more confusing, patients on thyroid replacement are considered taking "too much," and their doses are reduced inappropriately.[237-238]

Almost all patients are fatigued and most have some combination of polydipsia/polyuria/nocturia. Abnormalities in ADH/osmolality and ACTH/cortisol feedback loops are also usually demonstrated.

Histamine is a known messenger of dehydration, signaling ADH to correct the problem. The symptoms of sneezing, itching, and runny nose are often assumed by patients—not to mention healthcare providers—to be allergy, but with the depletion of ADH, there is a never-ending failure to end dehydration, and many patients suffer from fungal rhinitis, sinusitis, or rhinosinusitis. Since histamine is also alerting, there is a never-ending stimulus toward agitation and anxiety. Further, histamine is methylated to its inactivated state. Depleted methylation resources then result in a series of symptom presentations that resemble depression.[239-243] MSH is an infundibular pituitary hormone and levels are low in roughly 90% of cases.

Food-sourced histamine is frequently associated with histamine intolerance. The postulated mechanism is depletion of the enzyme Diamine Oxidase (DAO) in the GI tract. In an animal study, piglets were exposed to ingested lipopolysaccharide (LPS) endotoxin, and DAO levels were reduced. Animals that were either simultaneously or subsequently treated with L-arginine did not have a reduction in DAO.[243-246] LPS is a metabolic product of Gram negative bacteria which are often part of the mixed microbial environment in WDB.



A fourth mechanism of increased histamine results from mast cell activation. According to Theoharides, there is upregulation of mast cells by Corticotropin Releasing Hormone (CRH) and substance P (which is released at pain sites).[247-249] The number of receptors on mast cells for substance P further increases as the amount of substance P is raised.

Chronic production of pro-inflammatory cytokines with decreased beta-endorphins and rare restorative sleep lead to recurrent diffuse debilitating myalgias in many patients. Beta-endorphin is cleaved from Pro-opiomelanocortin (POMC). Since POMC is low, beta-endorphin is low. These symptoms are identical to those in many patients diagnosed with fibromyalgia.

There are no biomarkers for fibromyalgia, but there are 10 for this illness. As such, all patients diagnosed with fibromyalgia should be evaluated for this illness as a treatable cause of the myalgias.

Multiple derangements in regulation and the immune system can lead to autoantibody production and multi-system dysfunction.[250] Patients often develop anticardiolipin antibodies and can look very much like those diagnosed with systemic lupus erythematosus (SLE). In fact, this illness should be in the differential diagnosis of all patients previously labeled as SLE who do not demonstrate a positive anti-double stranded DNA antibody.

Clinicians treating this illness in patients have also observed a high rate of ANA positivity without diagnosable rheumatologic disease. There is a further connection here, in that the inability to recycle homocysteine to methionine and around, results in reduced production of SAMe. The effect is not only on methylation, but on the failure of SAMe (S-adenosylmethionine) to donate methionine to putrescine, which is derived from ornithine. Putresicine levels are elevated in Lupus. Putrescine + SAMe = Spermadine + SAMe = Spermine, which is the precursor to polyamines that encourage nerve healing.

Small vessel dysfunction is commonly seen in persons with this illness. There is also microvascular sludging, which is further associated with the hypercoaguable phenomena already described, capillary shunting, and increased venous oxygen, and excess intrinsic production of carbon monoxide due to the action of hemoxygenase on hemoglobin. This could be the result of persistent triggering of the innate immune system leading to white cell demargination which congests distal small arterioles and chronically decreases red blood cell delivery of oxygen to peripheral capillary beds as well as autonomic nervous system disturbance resulting from long term exposure to toxins.

Very cold hands and feet are frequent in sufferers of this illness and some patients report transient blue and even green color changes to appendages not consistent with Reynaud's phenomenon. Increased blood levels of erythropoietin and/or Vascular Endothelial Growth Factor (VEGF) abnormalities document the peripheral hypoperfusion as does MR spectroscopy of the brain.

In a 2014 study, statistically significant differences in brain structure proportions were seen for patients in both hemispheres of two of the eleven brain regions analyzed; atrophy of the caudate nucleus and enlargement of the pallidum.[212]

In addition, the left amygdala and right forebrain were also enlarged. These volumetric abnormalities, in conjunction with concurrent abnormalities in inflammatory markers, suggest a model for structural brain injury in 'mold illness' based on increased permeability of the blood-brain barrier due to chronic, systemic inflammation.[212,216]

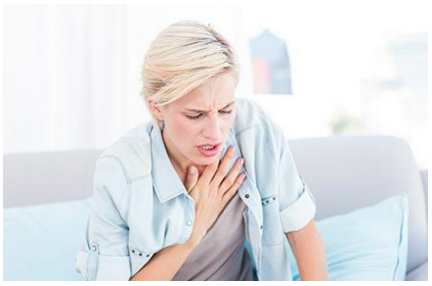
Shortness of breath is seen frequently in those exposed to water-damaged buildings. This can result from a variety of factors, including reactive airway disease, with evidence of small airway obstruction frequently noted in pulmonary function testing.[251-263]

The most common abnormalities are seen in markers of small airway obstruction such as $FEF_{25-75\%}$ (forced expiratory flow between 25 and 75% of forced vital capacity) and $FEF_{75\%}$. There is sometimes a restrictive component – which the pulmonologists will call "idiopathic."

When Autonomic Nervous System and Respiration (ANSAR) testing of heart rate variability is simultaneously assessed, this restrictive component is associated with decreased parasympathetic nervous system (PSNS) activity.

Since PSNS (vagus) regulates diaphragm contraction, there is typically an attempt at increased sympathetic nervous system (SNS) activity (chest wall expansion), to compensate for the decreased air entry. There is sometimes a net result of decreased FVC (forced vital capacity).[264-265]

We observed a differential association between mold and other aeroallergen sensitization, and severity of asthma. Sensitization to mold is associated with lower lung function and increased airway hyper-responsiveness in children with asthma.[265]



Additionally, pulmonary infections such as chronic *Mycobacteria*, including *Mycobacterium avium intracellulare* infections, can result from exposure to water-damaged buildings resulting in a variety of chronic pulmonary symptoms including shortness of breath.[53,136,266-270]

Hypersensitivity pneumonitis, allergic bronchopulmonary aspergillosis, bronchiectasis and, in severe cases, pulmonary fibrosis can result from exposure to water-damaged buildings [211,271-276] and mold. Low VEGF has also been demonstrated by some researchers to be associated with shortness of breath (even in well-conditioned athletes).

Neurologic and cognitive symptoms are some of the most frequent complaints, and typically is the most upsetting complex of symptoms experienced, in those exposed to water-damaged buildings.

Direct neurotoxic affects from mycotoxins and other toxins found in WDB, vascular hypoperfusion, cytokines and immune system inflammation after T_h17 driven breach of the blood brain barrier can all contribute to these phenomena.

Brain fog, disturbances in memory, concentration, balance, word finding difficulties and other cognitive symptoms are frequently seen in those exposed to water-damaged buildings.

What is not in dispute is that brain fog, disturbances in memory, concentration, balance, word finding difficulties and other cognitive symptoms are frequently seen in those exposed to water-damaged buildings, and a dose response relationship has been confirmed.[36-38,115,124,221,266,277]

Renal effects have also been seen with exposure to water damaged buildings. Ochratoxin A has been associated with Balkan Endemic Nephropathy in humans, urinary tract cancers in animals and humans and focal segmental glomerulosclerosis.[278-281]

Negative effects on eyes and skin also occur.[144-153] We discussed dermal and ocular effects in the previous section on Pathophysiology.

Ears can also be affected by fungi.[282-288] Tinnitus (ringing in the ears) is often found in patients exposed to water-damaged buildings. Patients are also often diagnosed with Meniere's disease.

In a 2016 study in Tanzania after severe flooding, they found that 97.1% of the homes had mold contamination.

> Five types of moulds were found dominated by a black spore former *Aspergillus niger* found in 87 houses (41.2%) followed by *Penicillium* species in 65 houses (37.1%) and *Cladosporium* species found in 60 houses (34.3%). The revealed moulds are well known to be associated with human health problems including production of carcinogenic metabolites, triggering allergic reactions to sensitive individuals, causing keratitis, skin lesions, nail fungus, sinusitis, intrinsic asthma, and pulmonary infections.[288]

Chronic fatigue is the overriding symptom which unites almost every patient with this illness although about 5% of patients do not describe fatigue as a recurrent symptom. Fatigue most likely results from chronic non-restorative sleep, low cortisol with dysregulation of the Hypothalamic-Pituitary Axis (HPA), peripheral hypoperfusion and mitochondrial injury resulting from exposure to the many toxins, such as mycotoxins, found in water-damaged buildings.[17-18,194,286,289-292]

It is important to note that pets (cats and dogs) have also been affected by exposure to contaminants in water-damaged buildings. There have been many reports regarding family pets that have become ill or died in water-damaged homes.

A 2012 study reported on a family of five and their pet dog who rented a water-damaged home and developed serious multiple health problems.

> The dog developed 72 cutaneous lesions that were distributed over its body, including the ears. The dog's urine was positive for ochratoxin A and trichothecenes. In addition, surgical specimens of the ear (sebaceous gland) and body tumors (lipomas) were also positive for trichothecenes and ochratoxin A.[36]

As reported in a national magazine for livestock farmers:

> Toxins produced exclusively by fungi are called mycotoxins and are fungal metabolites which when ingested, inhaled or absorbed through the skin reduces performance, sickness or death in man or animals, including birds.[293]

Later in this paper, in the section on Causation, we will provide information about studies that have been done on the inhalation effects of mycotoxins on mice, rat, guinea pigs and swine (pigs).

Diagnosis

Diagnosis of this illness is usually straightforward although a consensus of the exact definition of the disease has not yet been established. There are numerous objective biological indicators found in patients suffering from this illness.

The process of establishing a unique constellation of symptoms and lab findings is commonly used in medicine to delineate a diagnosis. The Jones criteria for Rheumatic Fever and the diagnoses of SLE and Kawasaki Syndrome are just three such examples.

Dr. Ritchie Shoemaker, et al, coined the term "CIRS-WDB" in 2008 and have proposed a three-tiered case definition. He and his group use history, physical exam findings, and results of Visual Contrast Sensitivity (VCS) testing, MR Spectroscopy, nasal culture and blood tests to look at ten different biomarkers for CIRS-WDB.[17-18,23,33,35,158,192-195,236] Healthy persons should have, on average, 5% of these markers positive (0-1 per patient) whereas cases usually manifest at least 5-6 (50-60%) abnormal values.[236,294-298]

It is expected that most CIRS-WDB patients will also demonstrate abnormal TGF-β1, VIP and/or C4a and the presence of MARCoNS. Many will show altered von Willebrand's, iron and/or androgen studies, and often reveal antibodies to cardiolipin and/or gluten (with negative tissue transglutaminase or TTG). MR Spectroscopy often reveals specific abnormalities such as increased glutamate to glutamine ratios. Careful attention to confounding diagnoses is also required.

In May 2018, Dr. Shoemaker and six of his associates released a Consensus Statement regarding the diagnostic process for CIRS. The information expands on previous papers regarding CIRS. Table 1 in the paper lists a percentage of incidence of symptoms that are common in CIRS patients. They grouped those symptoms into 13 clusters. If a patient has symptoms in at least 8 of those clusters (or 6 clusters for pediatric patients), there is adequate support for a CIRS diagnosis. They specifically state that the likelihood of CIRS exceeds 95% if the adult patient has 8 or more clusters. This new paper also provides specific details about the physical exam of CIRS patients.[236]

Other treating physicians use additional testing modalities.[299-302] Dr. Michael Gray *et al* use the term Mixed Mold Mycotoxicosis and also look for evidence of fungal colonization in nasal passages, sputum and stool, evaluate potential pesticide exposures and measure urine mycotoxins as proof of exposure. His group also looks at Nerve Conduction Velocities, neurobehavioral testing developed by Dr. Kaye Kilburn (found to demonstrate evidence of chemical brain injury in those exposed to environmental toxins including mycotoxins) and Quantitative Electroencephalograms (QEEG) as part of their evaluation.[29,36,111,115,266]

As noted earlier in this paper, in 2017, Dr. Michael Gray presented a new name for illness caused by exposure to toxins/ poisons which includes illness caused by many contaminants in our environment. He refers to it as Cumulative Organic Chemical Hyper-Toxicity.

In addition to much of the testing used by Dr. Gray, Dr. Janette Hope uses detoxigenomic studies which look at various single nucleotide polymorphisms (SNP) and assesses for nutritional deficiencies and food allergies frequently found in those with long-term toxic exposures.[34]

Dr. Alan Vinitsky assesses for autonomic nervous system (ANS) dysfunction via the Autonomic Nervous System and Respiration (ANSAR) testing system. He has developed a working model of hypomethylation to account for some of the symptoms of ANS dysregulation as they relate to stress. His treatments initiate recognition and correction of the dehydration patterns that relate to the ADH – Histamine reactions described above. In addition, he has identified a pattern of amino acid deficiencies that result in chronically ill individuals.[303-305]

By definition, all patients with inflammation have localized swelling, resulting in misplaced fluid in the body. The more extensive the inflammation, the greater is the fluid shift. Dr. Vinitsky has coined this condition "dyshydration©." And, the messenger is still histamine. Every person with water-damaged environmental illness should be queried for fluid consumption, and, in particular, water and minerals. When asked, many patients when advised that they are "dehydrated, they respond with: "But doc, I am already drinking a lot." Dyshydration© and an explanation of inflammation helps affected patients understand.

Patients with inflammation have localized swelling, resulting in misplaced fluid in the body. The more extensive the inflammation, the greater is the fluid shift. Dr. Vinitsky has coined this condition "dyshydration."©

Another aspect of histamine excess comes from the food patients consume. Food-based histamine is metabolized by the enzyme DAO (diamine oxidase). DAO may be reduced by LPS (lipopolysaccharide) or endotoxin. In an animal model, LPS was given to piglets, and DAO levels were reduced. When the same animals were simultaneously treated with arginine, DAO was not reduced. And, when animals were sequentially treated with LPS, followed by arginine, DAO was intact.

The pattern of 24-hour urine for amino acid deficiencies was phosphoethanolamine, hydroxyproline, asparagine and isoleucine. Sometimes glutamine and methionine are also depleted. Dr. Vinitsky's protocol on balance testing during physical examination is described. The loss of balance with eyes closed reflects mid-brain dysfunction. Autonomic function is separated from visual compensation in maintaining balance. Dietary L-arginine supplementation improves intestinal function in weaned pigs after an *Escherichia coli* lipopolysaccharide challenge.[243-246]

Significantly, in water-damaged environments of mixed microbes, Gram negative organisms (associated with LPS) often thrive. When inhaled or ingested by affected persons, LPS may then be the trigger for frequently observed food-induced histamine intolerance.

Dr. William Rea, one of the pioneers in the field of environmental medicine, has developed a multi-disciplinary approach to diagnosis (which includes intradermal provocation of mycotoxins) in a facility using state of the art construction techniques to create a "less polluted environment for patient evaluation, testing and treatment."[306-314] His group has suggested the moniker "Indoor Mold Sensitivity and Toxicity" for the disease.

Dr. Allan Lieberman's diagnostic protocol is similar to those mentioned above. After a careful medical and environmental history, he has a series of tests that are ordered depending on the history and symptoms of each patient.[40,113,120,315] Some of the tests included:

1) A basic metabolic panel
2) Measurement of antibodies to molds and mycotoxins in serum
3) Immune tests for autoantibodies, complement, gamma globulins and lymphocyte panels
4) Urine and blood testing for mycotoxins
5) Visual contrast sensitivity tests
6) Pupillometry and heart rate variation to assist in the evaluation of autonomic nervous system function
7) Standard neuropsychological test batteries
8) EEG and brain imaging techniques
9) SPECT and magnetic resonance imaging (MRI)
10) Pulmonary function tests

Two other pioneers in this field are discussed on the following pages, and the number of doctors and other medical professionals who know how to diagnose and treat this illness is increasing. You can find a comprehensive list of current treating physicians, along with their contact information, on the Paradigm Change website at paradigmchange.me/practitioners.

Treatment Protocols

Treatment protocols also vary and to date there have been no head-to-head trials on the efficacy or superiority of any one regimen. However, each listed practitioner will relate extraordinary results (even up to 90%) of patients who are compliant with the prescribed therapy. The two basic principles of most approaches include 1) toxin avoidance and 2) removal of toxin from the body—usually via sequestering agents.

There have been several influential treating doctors in this field. Two of the early pioneers were Dr. Kaye Kilburn and Dr. Vincent Marinkovich. Their contributions were significant and are briefly described in the following paragraphs.

Dr. Kaye Kilburn investigated asbestosis, byssinosis, mold, mycotoxins and hydrogen sulfide. He conducted one of the most extensive studies into asbestos, helping to expose its

danger in an industrial setting. His work with cotton dust helped set the modern standard for respiratory care and testing.[316]

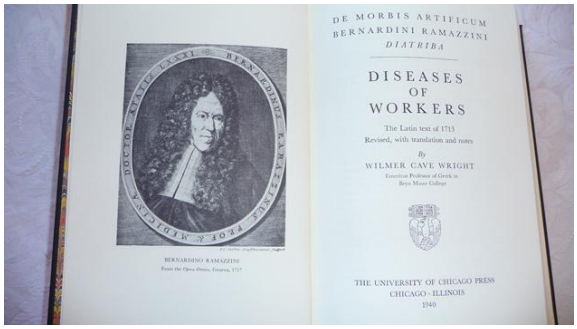
In 1958, Dr. Kilburn developed a cardiopulmonary research laboratory at the U.S. Army Medical Research and Nutritional Laboratory at Fitzsimmons Army Hospital in Denver, Colorado. In 1963, he established a new and novel division of Environmental Medicine at Duke Hospital.[316]

His work was included in a 1986 U.S. Surgeon General's Report that helped changed the way the nation views tobacco use---leading to warnings on cigarette packages and other legislation to curb the damaging effects of smoking.[316]

Dr. Kilburn was one of the earliest members of the Collegium Ramazzini, elected in 1983.[316]

The Collegium Ramazzini is an independent, international academy founded in 1982 by Irving J. Selikoff, Cesare Maltoni and other eminent scientists. It is comprised of 180 internationally renowned experts in the fields of occupational and environmental health. The mission of the Collegium Ramazzini is to advance the study of occupational and environmental health issues and to be a bridge between the world of scientific discovery and the social and political centers which must act on the discoveries of science to protect public health.

The Collegium Ramazzini was named in honor of Dr. Bernardino Ramizzini. He created the science of occupational medicine in the 17th Century in Italy. Dr. Ramizzini demonstrated the importance of talking directly with workers and of visiting workplaces to investigate the working environment in order to improve it. He focused on the need for providing workers with adequate information about health hazards, and he suggested practical measures to protect workers from illness and injury.[317]



In our paper titled "Indoor Air Contaminants," we refer to the renowned book written by Dr. Ramizzini in 1700 titled *De Morbis Artificum (Diseases of Workers)*. It discusses the health hazards affecting workers in 40 different occupations, including chemicals, dust, metals and other agents.

In 1987, Dr. Kilburn founded his own practice, Neuro-Test Inc., to study neurobehavioral and pulmonary impairment as a result of exposure to common chemicals including mold, hydrogen sulfide, diesel, pesticides and insecticides.[316]

In regard to patients exposed to molds, mycotoxins and chemicals, Dr. Kilburn demonstrated the benefits of neurocognitive testing as these toxins cross the blood brain barrier.[22,36,42,54,318-319] In his treatment of patients, he used many of the methods described in this section and also used Dr. Grace Ziem's oxidative stress protocol (Neural Sensitization Protocol or NSP) which targets the increased oxidative stress associated with both inflammation and toxicity.[320]

As Dr. Kilburn described in a 2009 paper:

> Colonization of nasal sinuses and local extension; tissue destruction and swelling, coupled with systemic toxicity produce extreme fatigue, headache, hearing loss, joint pain, tremors, depression, and direct central nervous system effects with abnormal balance, loss of concentration and of verbal recall, and long-term memory.[22]

> Poisoning by inhaling mycotoxins can explain multi-organ symptoms and neurological impairment for balance, reaction time, muscle strength, color discrimination, visual field performance, hearing, and blink reflex latency. Cognitive performance, verbal recall, and long-term memory are impaired, especially the ability to recognize missing items in standard pictures (Kilburn, 2009). This pattern is identical to that after exposure to H2S, chlorine, solvents, and other single toxic chemicals that are small molecules.[22]

This paper goes on to explain the evolution of this problem, including the use of gypsum wallboard/drywall (which is a food source for mold) to build homes and schools after World War II. He also discusses the transport of spores across the planet including the death of coral reefs in the Caribbean and a significant increase in asthma caused by mold traced to the deserts of Africa and Asia borne in trade winds.

> Crossing the country with other pollutants like lead and mercury, the isotopes of which show the African origin of the dust spores, that land everywhere.[22]

Dr. Kilburn published more than 250 research papers during his career. He passed away in 2014.

Dr. Vincent Marinkovich specialized in diagnosing and treating mysterious ailments caused by household molds.[71,81] He was known as Dr. Mold, and he continued his devotion to help his patients until he passed away in 2007.

In 1976, Dr. Marinkovich developed the MAST allergy blood test (Multiple Allergen Simultaneous Test) that detected allergens with the use of cellulose fibers in an enzymatic test chamber.

> The MAST Immunodiagnostic Test System was developed to provide a comprehensive, simple means for the in vitro measurement of multiple antigens or antibodies. The MAST system greatly simplifies testing for allergen-specific IgE, while retaining specificity and sensitivity.[321]

In a 2004 paper by Dr. Marinkovich, he discussed the effects of mold on the human body and the diagnosis, pathophysiology and therapy/treatment. He also talked about the doctors who refuse to recognize this illness, as follows:

> There are other physicians who deny that fungi as encountered in homes or office-type workspaces are capable of causing illness. These physicians generally are not primary caregivers and can dismiss the patient's complaints because of their apparent complexity without a consequence. They are better designated as theorists who base their negativity on arguments that the lack of sufficient evidence-based proof of a causal relationship of fungal exposure to human disease proves that such a relationship is not possible.
>
> They dismiss all case reports, epidemiological studies and clinical observations of experienced clinicians as worthless. They seem to lack the vision to accept the challenge of the possibility that injury to multiple organ systems may result from exposure to large amounts of fungal derived materials (such as spores and/or mycotoxins) in a home or office environment.
>
> They are wrong and they can do a great deal of harm. First in denying the patient's symptoms, and secondly by blocking disability requests from such patients injured by exposure to fungi in their workplaces. They are guilty of using poor scientific logic because it is close minded. Such thinking has no place in a medical setting where there are sick patients who need help.[71]

There are other physicians who deny that fungi encountered in homes or workspaces are capable of causing illness.

They seem to lack the vision to accept the challenge of the possibility that injury to multiple organ systems may result from exposure to large amounts of fungal derived materials (such as spores and/or mycotoxins) in a home or office environment.

They are wrong and they can do a great deal of harm.

Dr. Claudia Miller first introduced the theory of TILT (Toxicant Induced Loss of Tolerance) in 1996. Her work with patients with chemical intolerance (also referred to as chemical sensitivity) led to the development of the Quick Environmental Exposure and Sensitivity Inventory (QEESI). The QEESI test is used for screening patients with multiple chemical intolerance.[322-325]

In 2012, Dr. Miller proposed a new level of "LEED Diamond" for LEED-certified buildings. Although builders can earn extra points for meeting certain requirements for improving indoor air quality (IAQ) under the current LEED categories (silver, gold and platinum), additional requirements relating to IAQ are needed. As Dr. Miller said:

> Someday, we will look back at how we constructed and operated our buildings and realize that we should have paid far more attention to IAQ (indoor air quality). At that time, it will be clear what we should have done long ago–-designing buildings for the most vulnerable individuals in our population (about 1/3 of the population). Protecting the most susceptible people will protect everyone.[326]

Discussion of Treatment Protocols

As mentioned above, the two basic principles of most treatment approaches include 1) toxin avoidance and 2) removal of toxin from the body—usually via sequestering agents. The following discussion explains some of the other treatment methods used by the leading physicians in this field.

All of the treating physicians in this field recommend avoiding mold exposure as a key component of their treatment plan. They also use various sequestration methods including cholestyramine, bentonite or zeolite clay and/or activated charcoal. The clay and cholestyramine are mixed together in a liter of water and drunk over the course of the morning/afternoon and then again after dinner. The charcoal is taken as pills or capsules.

Some of these treating physicians use glutathione and targeted nutritional support to promote detoxification, as well as exercise and sauna therapy when indicated. Additionally, some physicians recommend using food-grade diatomaceous earth (which is made of silica). Silica is sometimes used as it provides an additional benefit by trapping metal toxins, biotoxins and pesticides.

The following discussion provides additional details about the various treatment protocols.

Beyond toxin avoidance and sequestration, Dr. Ritchie Shoemaker and doctors who have completed the certification process for Dr. Shoemaker's treatment protocol for CIRS (Chronic Inflammatory Response Syndrome, also known as CIRS-WDB), follow a 14-step, pyramidal approach to therapy.[236,297] As each step is cleared, more patients will be free of symptoms and have a return to the normal biologic regulation and lab work which healthy persons enjoy.

The first doctor to complete Dr. Shoemaker's certification process is Dr. Scott McMahon in New Mexico. He has worked closely with Dr. Shoemaker and has authored or co-authored research papers on this topic.[193,216,236,327]

Another doctor who completed Dr. Shoemaker's certification process is Dr. Mary Ackerley, an integrative psychiatrist. She follows the Shoemaker protocol with her patients. She also focuses on the inflammatory effects on the brain. She cites research studies documenting cognitive impairments, decreased executive functioning, depression and suicidal thoughts in patients who have been exposed to contaminants in water-damaged buildings and has seen this in her own patients.[214]Our list of references includes several papers on the neurological effects of molds, mycotoxins and other contaminants in water-damaged buildings.

Dr. Michael Gray also stresses the need for adequate upper respiratory and pulmonary care, uses supplements such as CoQ10 and, on occasion, enlists systemic anti-fungal agents.[29,36,111,115,266,328] Glutathione is heavily emphasized in Dr. Gray's treatment protocol, being used in an oral liposomal form, nebulized and intranasally.

Dr. Janette Hope, in addition to the use of sequestering agents (cholestyramine and charcoal), also prescribes glutathione via all of the above routes and nasal antifungals when indicated, treats detoxigenomics findings to specifically address genetic deficits (SNPs) and nutritional testing to assess for adequate presence of vitamin cofactors needed for proper detoxification. Treatment includes both avoidance of problematic medications, toxins, foods and hormones as well as supplementation of specific cofactors (magnesium, B vitamins, etc.)[34,278]

Dr. William Rea has treated more than 30,000 patients over the past 40 years. He has developed a comprehensive protocol for treatment and diagnosis. Dr. Rea's approach is aimed at decreasing the "total body load" of all toxins and toxic chemicals, injections to neutralize mycotoxins, avoidance of foods and chemicals to which patients may have become sensitized, parenteral and oral nutrition (the latter includes spring water in glass bottles, organic foods and a rotary diet), sauna treatments, exercise and massage. Some patients require an autologous lymphocytic factor, developed at Dr. Rea's center, which modulates the patient's own immune system. In some others, anti-fungals, oxygen therapy and sequestration agents are used.[31,40,251,306-314]

In a 2016 paper published by Dr. Rea, he describes the "coherence phenomenon" and how it ties together exposures to mold, pollen, dust, food, chemicals and electromagnetic fields.[313] As mentioned above, he uses a less-polluted, controlled environment that provides more precise diagnosis and treatment. As he explains:

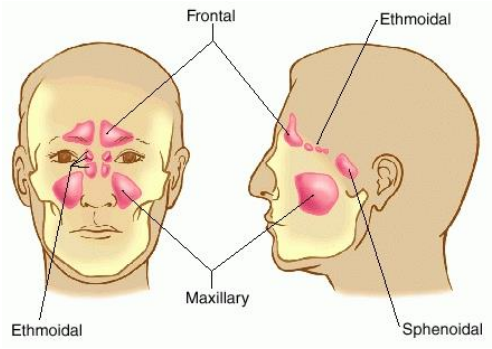
Dr. Rea describes the "coherence phenomenon" and how it ties together exposures to mold, pollen, dust, food, chemicals and electromagnetic fields.

> The principles of diagnosis and treatment depend on total environmental and total body pollutant loads, masking or adaptation, bipolarity of response, and biochemical individuality, among others. These principles make less-polluted, controlled conditions necessary.[313]

Dr. Rea has presented several papers on environmentally triggered cardiovascular disease, vasculitis, phlebitis, including biopsies, incitant tests and immune parameters, and implants, showing toxic substances could cause internal problems like gastrointestinal (GI) and genitourinary (GU) malfunction, kidney disease, and cardiovascular and brain dysfunction, in addition to fatigue, fibromyalgia, and ENT disease.[306-314]

Dr. Walter Hayhurst and Dr. Donald Dennis suggest treating fungal infections in the sinuses aggressively[329] and use "The Inflammation Free Diet Plan"[330] and recommend resveratrol, a molecule shown in some studies to prolong the lifespan of worms, fruit flies and short-lived fish.[331-332] It may also reduce the risk of certain cancers in rats. Additional studies

have been completed regarding the neuroprotective, anti-inflammatory, cardioprotective, anti-diabetic and anti-viral effects of resveratrol.[333-335]



Dr. Dennis has also authored research papers on case studies involving fungal sinusitis.[36,287,329,336] In a 2016 report, he took a swab of the mass in the patient's left ethmoid sinus which showed bacteria, candida and ten other types of fungi.[287] In that report and again in a 2017 report, he discusses the surgical and medical management of sinus mucosal and systemic mycotoxicosis.[336] The tissue samples from both patients tested positive for mycotoxins.

Dr. Alan Vinitsky teaches that chronic overstimulation of the sympathetic nervous system (SNS) also contributes to "mold toxicity." His therapies also include relaxation/meditation techniques, energy optimization, dietary changes, exercise, nutritional supplements, increasing purified water intake and development of a positive mental attitude to help the body heal itself of toxins and toxic stress as precursors to the inflammatory response.[303-305] The intent is to integrate the mind, body and spirit into the healing process.

As a further metabolic basis of treatment, Hydroxocobalamin is a recognized scavenger of inflammation stress (nitric oxide) as described by Martin Pall.[337] That is part of the basis of Dr. Ziem's protocol.

Dr. Vinitsky has identified a 5:2 ratio of sublingual, transbuccal or intravenous Hydroxocobalamin Folate as a means of cleaning up oxidative stress (aldehydes) and Nitric Oxide -- first as a means of cleaning up stress, then replenishing sufficient doses on a patient-to-patient basis to correct methylation defects. He has defined the Methylation Priority Principle© as a relative ranking of the need for methylation processes, of which there are more than 50 in the body. Included in this ranking are Adrenalin− activation and inactivation; followed by Histamine, Metals and Estrogen; then neurotransmitters Norepinephrine, Dopamine, Serotonin, and Melatonin; next RNA synthesis and DNA, histone, and microRNA repair; and finally, creatine production. In a recent review of 8 years of using this open-label protocol for patients of all ages, a significant portion who have been mold-affected, more than 2.37 million doses have been administered.[303]

Dr. Joseph Brewer follows a treatment protocol similar to the other doctors mentioned in this section. He has published research on the connection between mycotoxins and chronic fatigue syndrome and sinusitis.[289,338] Mycotoxin testing of urine samples has been discussed in other studies.[289,328,339]

Dr. Irene Grant has worked closely with Dr. Jack Thrasher and others in this field, and she also follows a similar treatment protocol.[340-342]

Dr. Raymond Singer, a Ph.D. neuropsychologist, and Dr. Robert Crago, a Ph.D. psychologist, provide testing and evaluation of patients with neurological damage and brain injuries caused by mold, chemicals and other toxins.[29,115,119,141,221,226,343-345]

As mentioned earlier in this paper, the number of doctors and other medical professionals who know how to treat this illness is increasing. You can find a list, along with their contact information, on the Paradigm Change website at paradigmchange.me/practitioners.

Mold Avoidance and Testing

Toxin avoidance is another important component of treatment for many patients. A discussion of tips and techniques on mold avoidance is presented in a book titled "A Beginner's Guide to Mold Avoidance: Techniques Used by Hundreds of Chronic Multisystem Illness Sufferers to Improve their Health."[346] Additional tips about mold avoidance are provided in a book titled "Erik on Avoidance: Writings about Mold Avoidance."[347]

Avoidance of mold and other toxins is an important component of treatment for many patients with this illness.

In addition to following mold avoidance, Dr. Shoemaker suggests testing suspected indoor spaces via ERMI which detects mold DNA from vacuumed carpet or flooring samples. The result is logarithmic and a score of 2.0 or less is considered acceptable unless the patient has low MSH or very high C4a lab values.

Unfortunately, there continue to be limits to most testing modalities, and it is often necessary to evaluate indoor settings using historical information about the building, as well as signs of water damage and moisture excess, combined with judicious use of focused testing. Multiple testing modalities exist and most experts agree a combination of methods provides optimal results.[348-352]

The following testing methods are recommended by Dr. Jack Thrasher, a world-renowned toxicologist in this field.[29,36-38,52-53,336,338,340] Prior to doing any tests, a thorough investigation and evaluation should be conducted. Dr. Thrasher provides the following list of key points to consider when testing indoor environments. This list is not intended to provide a complete discussion of this complex topic.

1) Air sampling for fungi and bacteria, under two different conditions:

    a. (1) Viable spore traps that can be counted for spores per cubic meter and cultured for Gram negative and positive bacteria. This is done as the home is normally occupied.

    b. (2) Use a compressed sterile air canister and disturb the indoor environment causing settled dust to be re-suspended. Perform the counts and cultures as described in (1) above.

You must be aware that air sampling for mold spores is not very reliable. It only tells what is in the indoor air at the time of sampling. It does not necessarily identify all mold because some growth can be hidden in wall spaces, attics and other areas.

2) RealTime PCR-DNA on bulk and dust samples

It is recommended that you collect dust from areas that do not normally get cleaned, such as on top of kitchen cabinets, behind washers and dryers, grates on the backside of the refrigerator, HVAC units, ducts and vents, under refrigerators and on the coils and compressors, the underside of carpets, etc. Bulk samples should be taken of visible mold growth.

3) Bacteria

Both Gram negative and positive bacteria grow along with the fungi.

Gram negative bacteria are potential human pathogens causing respiratory and other infections. Culture on blood agar plates at 37 oC and 50 oC. Thermophilic bacteria will grow at 50 oC and will be crowded out at 37 oC. Thermophilic Gram negative bacteria are also potential human pathogens.

Gram positive bacteria include *Bacillus* species, *Streptococcus*, *Staphylococcus*, *Micrococcus* and several different *Actinomycetes*. The *Actinomycetes* consist of several genera including *Streptomyces*, *non-tuberculin Mycobacterium*, *Nocardiosis* and a few other genera. Cultures for these organisms should be at 37 and 50 oC.

4) Endotoxins and 1-3-beta-D-glucans

Endotoxins are lipopolysaccharides that are present in the cell wall of Gram negative bacteria. They are known to be synergistic to the toxic effects of mycotoxins.

1-3-beta-D-glucan (glucans) are polysaccharide components of the cell wall of fungi.

5) Mycotoxins

Mycotoxins are produced by almost every species of fungi present in the indoor environment.

6) Particles

Colonies of fungi and bacteria shed particles (particulates). The particles are shed because of vibrations set up by normal human activity (TV, radio, talking, walking

and air conditioning currents) as well as by opening and closing doors. The particles range from less than one micron up to the size of fungal spores. Therefore, particle counts should be done by a meter that detects particles from less than one micron up to what is referred to as PM10. Be sure you ask the company you have selected if they can do this testing.

7) Moisture

Moisture is the most important factor that permits fungal and bacterial growth. Be sure that you have the home or building tested for moisture content using appropriate moisture meters that read out. Take photos of the readings. Be sure that moisture content of walls, flooring and other areas is measured. Should readings indicate that a wall cavity is positive for moisture, the wall cavity must be tested for fungi and bacteria.

8) Hidden water intrusion, mold and bacteria and their toxins

Dr. Thrasher strongly advises careful and thorough evaluation and investigation of all possible points of water intrusion. He provides the following example:

This case highlights the moisture intrusion that was missed in previous inspections and remediation of a home where occupants had ongoing health problems.

A four-bedroom, newly-constructed home was tested using a moisture meter and thermography. The home had been inspected, tested and re-mediated twice before his involvement.

Moisture readings of the concrete slab revealed 36 areas of increased moisture. Removal of the carpeting and linoleum revealed cracks in the slab. Review of construction practices demonstrated that the contractor did not use metal reinforcement when pouring the concrete slab.

Moisture readings around all windows showed elevated moisture at the bottom sides of two windows. Removal of outside stucco showed that the aluminum frame of the window was bent when installed, allowing moisture intrusion.

Finally, moisture readings were elevated around the fireplace, which had previously been repaired. Removal of the stucco showed that the tar paper wrapping had been punctured at the time of construction. The carpenters had used nail guns and missed the 2 x 4 studs.

The original industrial hygienist found only a fireplace leakage and water damage in the master bath. Therefore, the two initial remediation efforts only took care of the initial fireplace and bathroom mold growth. The water intrusion via the concrete slab and the two windows was not identified.

9) Basements

If you own a home with a basement, water intrusion through the walls is a major problem leading to both fungal and bacterial contamination. This results because of the porous nature of the walls surrounding the basement and the fact that dirt is piled against the exterior of the walls with no water barrier.[348]

As mentioned above, testing for contaminants in water-damaged buildings is a very complex situation. Although many government agencies say that testing is not necessary, there are some situations where testing may be desired or needed. For example, the U.S. EPA says that you may consider sampling as part of your site evaluation in specific instances, such as:

- Cases where litigation is involved
- The source(s) of the mold contamination is unclear
- Health concerns are a problem[349]

If you do not have extensive experience and/or are in doubt about sampling, consult an experienced professional. This individual can help you decide if sampling for mold is useful and/or needed and will be able to carry out any necessary sampling.[349]

The U.S. Centers for Disease Control and Prevention (CDC) issued a report about mold prevention strategies and possible health effects in the aftermath of hurricanes and major flood. Here are three brief excerpts from the report:

> In the aftermath of major hurricanes and floods, buildings or materials soaked for >48 hours are contaminated with mold unless proven otherwise by inspection or adequate environmental sampling or cleaned according to the EPAs recommendations.
>
> Although molds also might directly attack the skin or openings in the skin, the most common route of exposure is through the air and into the body by inhalation.
>
> Exposure to materials and structures contaminated with mold should be assumed to present a potential health risk regardless of the type of mold. Risk for illness does not necessarily vary with the type of mold or the extent of contamination.[69]

Another factor is the possibility of false-negative test results which are easy to obtain, especially using the common 5-minute spore trap techniques. However, it is nearly impossible to obtain a false-positive test result; therefore, all positive results should be taken seriously. Making sure home and work are mold-free places is critical for everyone.

Schools are a more challenging locale to test as the school district usually must give permission. Often, school districts and employers will not allow testing of their buildings.

Many government agencies state that testing is not necessary or not recommended or that exposure limits have not been set.[350-355] However, OEHCS has published books that discuss international exposure standards for mold, bacteria and chemicals.[356-357]

Decisions about testing depend on many independent and inter-dependent factors including whether the parties are involved in litigation and the current health status, sensitivity, and/or genetic susceptibility of each individual. An additional factor that needs to be considered is that testing is not financially viable for all homeowners, due to the extent of the damage and the tremendous financial losses that families incur in these situations. If testing is used or needed, positive results are a guide to treating the occupants of the exposed site, but negative results do not rule out the need to appropriately remediate.

In addition, a lack of moisture detection at the time of investigation may lead to a false sense of security. Mycotoxins are present in dust and can be on the hidden side of a "dry wall" which had previously been wet.

As noted above, a combination of testing modalities provides optimal results. Indoor air quality testing alone is often not sensitive enough to detect all possible contaminants (especially when some are hidden) or the low levels required to cause illness.

Dr. Thrasher worked tirelessly throughout his life to advance the science in the field of toxicology. He freely shared his knowledge and expertise with everyone. He published dozens of peer-reviewed research papers and case studies on the toxic effects of various chemicals, bacteria, molds and mycotoxins on animals and humans. He passed away in January 2017, but people from around the world continue to access his work and his research papers through his website (drthrasher.org).

Remediation

For spaces found to be "moldy" (i.e., water-damaged, regardless of the findings on testing, when used), remediation by certified personnel is recommended. Improper efforts can spread microbes (such as mold, bacteria and parasites), spores, fragments and toxins throughout the entire structure as water-damaged building materials are removed.

If remediation is attempted, proper containment procedures and personal protective equipment are critical, because disturbing or handling the contaminants can result in increased aerosolized spores and particles containing mycotoxins which can be dangerous to human health and destructive to property.[44,93,158,228,236,263,296,352-369]

Personal Protective Equipment (PPE)

During remediation or renovation of water-damaged structures, personal protective equipment (PPE) should be used to protect the occupants and workers.

- Protective clothing that covers the entire body (i.e., a disposable body suit such as TYVEK with mold-impervious, disposable head and foot coverings). All gaps in the clothing, such as those around ankles and wrists, should be sealed.
- An N-95 respirator or better (i.e., a half-face or full-face respirator with N, R or P100 filters)
- Protective gloves (made of natural rubber, neoprene, nitrile, polyurethane or polyvinylchloride). Do not touch mold or moldy items with bare hands.
- Non-vented goggles. Wear goggles that provide complete eye protection. Choose goggles designed to keep out dust and small particles. Safety glasses or goggles that have open vent holes will not protect you against dust and small particles.[69,276,349,359,363,365-369]

During remediation, proper containment procedures and personal protective equipment are critical, because disturbing or handling the contaminants can result in increased aerosolized spores and particles containing mycotoxins which can be dangerous to human health and destructive to property.

The U.S. Centers for Disease Control and Prevention (CDC) provides the following guidance regarding personal protective equipment (PPE):

> Primary functions of PPE in a mold-contaminated environment are prevention of the inhalation and ingestion of mold and mold spores and prevention of mold contact with skin or eyes.
>
> Respirators used to protect persons from airborne contaminants (including mold and mold spores) must be certified by CDC's NIOSH. In addition, as specified by the OSHA respiratory protection standard (37), workers whose employers require them to use respirators must be properly trained, have medical clearance, and be properly fit-tested before they use the respirator. Formal fit testing is recommended for anyone engaging in remediation work causing extensive exposure to mold.
>
> Persons doing remediation work that involves extensive exposure to mold should have respiratory protection greater than that provided by a NIOSH-certified N-95 respirator. Full face-piece respirators that have NIOSH-certified N100, R100, P100 particulate filters are recommended.[69]

The processes involved in accomplishing effective mold remediation are dependent upon multiple factors as each water damage situation presents its own unique set of circumstances and challenges. For example, because some water-damaged structures may produce a false negative test result based on sampling, remediation procedures should still be implemented.

It is important to note that remediation plans should not be prepared by the person or company who will be doing the remediation work. There needs to be a separation of duties in order to ensure proper procedures and industry guidelines are outlined in the remediation plan and to avoid any conflict of interest.

Mycotoxins routinely travel with spores (alive or dead) and, even more concerning, travel with very small, even submicron-sized particles capable of penetrating deep into the lungs. At this level, they are subjected to the effects of pulmonary surfactants which allow otherwise insoluble toxins to be absorbed into the bloodstream. And, as mentioned earlier in this paper, mycotoxins can cross the blood-brain barrier.[17-18,34,36,41,49,268-269,318-319,370-371]

A report by the U.S. Department of Housing and Urban Development (HUD) discusses molds and mycotoxins, as follows:

> Many molds are also known to produce mycotoxins, which are toxic metabolites that can be a health hazard to birds and mammals upon natural exposure, i.e., ingestion, dermal contact, or inhalation. While common outdoor molds present in ambient air, such as *Cladosporium cladosporioides* and *Alternaria alternata*, do not usually produce toxins, many other different mold species do. Genera producing fungi associated with wet buildings, such as *Aspergillus versicolor*, *Fusarium verticillioides*, *Penicillium aurantiogriseum*, and *Stachybotrys chartarum*, can produce potent toxins, measurable in mold mycelia, spores, and the matrix in which the mold is growing. A single mold species may produce several different toxins, and a given mycotoxin may be produced by more than one species of fungi. Furthermore, toxin-producing fungi do not necessarily produce mycotoxins under all growth conditions, with production being dependent on the substrate it is metabolizing, temperature, water content and humidity.[50]

Some experts suggest creating a "safe room" in a moldy dwelling for those who cannot afford to properly remediate the entire space and also thoroughly cleansing pets and vehicles with natural (non-chemical) products. Safe rooms are created by positively pressurizing a room and using HEPA air purifiers.[372-373]

It is important to state there are some water damage situations that cannot be resolved or corrected with remediation, and remediation is not always successful. As such, it is not practical to provide a detailed discussion of mold remediation in this paper.

The 2013 report from the U.S. National Institute of Occupational Safety and Health (NIOSH) provides good general advice: "Building owners and employers should always respond when occupant health concerns are reported."[44] The report also offers the following words of caution:

> Renovation (and remediation) projects can create the release of airborne dusts, microbiological contaminants, gasses, and odors from both inside and outside of a building. Therefore, careful planning is essential to prevent exposures to building occupants. Key factors to consider include scheduling projects during times of low or non-occupancy, isolating work areas from occupied areas using temporary barriers,

negative pressurization to prevent migration of air contaminants into occupied areas, and HEPA filtration.[44]

Inappropriate remediation (e.g., painting over water-damaged materials, adding air-fresheners in areas to mask musty odors, and applying disinfectants or biocides to damp or moldy surfaces) can cause further problems with building degradation and symptoms in occupants.[44]

Inappropriate remediation (e.g., painting over water-damaged materials, adding air-fresheners in areas to mask musty odors, and applying disinfectants or biocides to damp or moldy surfaces) can cause further problems with building degradation and symptoms in occupants.

In the 2012 study by Peitzsch et al,[361] researchers tested ten commonly used agents purporting to be capable of neutralizing mycotoxins and/or suppressing mold growth; not one of them completely removed all mold and toxins. The report states (in part):

> We compared the efficiency of some commercially available products and methods used for remediation of mould-contaminated building materials. Samples of gypsum board and pinewood were artificially contaminated with toxin-producing isolates of *Stachybotrys chartarum* and *Aspergillus versicolor*, respectively, then, ten different remediation treatments were applied according to the manufacturers' instructions. Microbial and chemical analyses of the infested materials were carried out both immediately before and after treatment, after six weeks of drying at room temperature, and after another six weeks of remoistening. The aim of the study was to determine whether the investigated methods could inhibit the mould growth and destroy some selected mycotoxins produced by the moulds. None of the decontamination methods tested could completely eliminate viable moulds. No remediation treatment eliminated all the toxins from the damaged materials. These results emphasize the importance to work preventatively with moisture safety throughout the construction processes and management to prevent mould growth on building materials.[361]

Another paper published in 2018 showed that employees in a large office building continued to have respiratory and non-respiratory effects even after multiple remediation attempts. They summarized their findings as follows:

> Our study indicates that once respiratory or severe non-respiratory symptoms have developed from long-term exposure to dampness and mold, the symptoms might not be easily improved despite various remediation activities. Our findings suggest that in moisture-damaged buildings with sentinel cases of building-related lung disease, the best public actions would be prompt relocation of affected employees, which might prevent further exacerbation of their illness or prompt remediation once water leaks are identified, that is before respiratory and severe non-respiratory symptoms have developed in building occupants.[102]

Building owners, homeowners and others responsible for the proper maintenance of structures are encouraged to contact experienced, knowledgeable and certified professionals for appropriate guidance. As mentioned above, it is not practical to provide a detailed discussion of mold remediation in this paper. However, we are going to provide a few brief comments about HVAC systems, ducts and biocides.

HVAC Systems and Ducts

HVAC (heating, ventilating, and air-conditioning) systems and ducts are common topics in regard to remediation of homes and buildings with mold contamination. As mentioned above, there are some situations that cannot be remediated. HVAC systems are difficult because there are many parts and because it is used to move air through the structure.

In addition, as the Peitzsch study concluded: "None of the decontamination methods tested could completely eliminate viable moulds. No remediation treatment eliminated all the toxins from the damaged materials."[361]

And, a 2018 study showed that occupants of a large office building were still having symptoms even though there had been repeated attempts to remediate.

> Our findings suggest that in moisture-damaged buildings with sentinel cases of building-related lung disease, the best public actions would be prompt relocation of affected employees, which might prevent further exacerbation of their illness or prompt remediation once water leaks are identified, that is before respiratory and severe non-respiratory symptoms have developed in building occupants.[102]

There are several papers and guidelines from government agencies regarding the remediation of HVAC systems. To give you an idea of the contaminants that can be found in HVAC systems,[52,353,374-376] the following description is from a 2015 paper regarding the investigation of dust loading and culturable microorganisms of HVAC systems in 24 office buildings.

> To investigate the dust loading and culturable microorganisms contamination characteristics of HVAC systems in 24 office buildings, a series of field tests, which included temperature, RH, air velocity, dust loading, culturable fungi/bacteria loading, were conducted in the following: return air, fresh air, mixture air, cooling, and supply air segments. There were significant positive correlations between dust and culturable fungi loading, dust and culturable bacteria loading, culturable fungi number per gram dust and RH, bacteria number per gram dust and temperature ($p < 0.05$). Results of these field measurements indicated that dust accumulation and/or high humidity and/or temperature should be properly controlled in HVAC systems to prevent the growth of culturable fungi and bacteria.[374]

A related topic is whether you should have the ducts cleaned. The U.S. EPA says you should consider having your HVAC ducts cleaned if 1) there is substantial visible mold growth,

2) ducts are infested with vermin, or 3) ducts are clogged with excessive amounts of dust and debris and/or particles are actually released into the home from your supply registers.[377]

The EPA also says that service providers should follow the air duct cleaning standards set by the National Air Duct Cleaners Association (NADCA).[378]

The NADCA says homeowners should consider having the HVAC ducts cleaned if 1) there are smokers in the household, 2) there are pets that shed high amounts of hair and dander, 3) there is water contamination or damage to the home or HVAC system, 4) there are residents with allergies or asthma, or 5) after home renovations or remodeling or 5) prior to occupancy of a new home.[378]

It is important to keep in mind that contamination is not just "visible" mold, but it also includes "invisible" particles that can be in the HVAC system which means they can be airborne throughout the building.

Biocides (including bleach)

Another common topic regarding remediation of water-damaged buildings is whether bleach or other biocides should be used. The answer is No.

Serious disinformation has been popularized and reflected in the guidelines given healthcare workers and the public encouraging the use of bleach (sodium hypochlorite) and other chlorinated products for cleaning the mold from damp indoor spaces. Biocides, including chlorine bleach, are harmful to humans and pets.[379-390] Bleach is a strong corrosive material and will irritate the eyes, skin and respiratory tract. It is cytotoxic and genotoxic and has an accumulative effect on the body and can cause damage to organs.

In regard to using bleach on mold, at least two species of concern—S*tachybotrys*—propagate via spores that are unaffected by chlorine, acids, caustics or ozone. In addition, chlorinating carbon-based organic toxins increases their toxicity by increasing their mutagenicity and their lipid solubility which allows these poisons to enter the skin and accumulate in lipid rich tissue such as fat deposits and the brain.

OSHA was the first U.S. government agency to remove the recommendation for using bleach on mold, and they acknowledge that biocides are toxic to animals and humans.[359]

Eventually, the EPA followed suit and said:

> Biocides are substances that can destroy living organisms. The use of a chemical or biocide that kills organisms such as mold (chlorine bleach, for example) is not recommended as a routine practice during mold cleanup.[388]

Even though the CDC acknowledges that bleach is not safe,[389] they still recommend bleach for removing mold growth from hard surfaces and HVAC systems.[390]

In regard to remediation of HVAC systems and ducts, the NADCA says source removal is the best method for cleaning HVAC systems. For best results, the entire HVAC system should be cleaned, including coils, blowers, and other components of the system.[378]

In regard to whether biocides should be used inside air ducts, the NADCA does not recommend the use of chemicals within ductwork unless there is a specific need. They state:

> Air duct cleaning service providers may tell you that they need to apply a chemical biocide to the inside of your ducts to kill bacteria (germs) and fungi (mold), and prevent future biological growth. Some duct cleaning service providers may propose to introduce ozone to kill biological contaminants. Ozone is a highly reactive gas that is regulated in the outside air as a lung irritant. However, there remains considerable controversy over the necessity and wisdom of introducing chemical biocides or ozone into the duct work.[387]

What about the claims of some service providers that they need to use chemicals to "sanitize" the ductwork? According to the NADCA, using biocides to sanitize ductwork is illegal. They state:

> The EPA has not registered any products for sanitizing or disinfecting ductwork. Further, no fungicides are registered for use in ductwork. It is a violation of federal law to use a product in a manner inconsistent with its labeling. For antimicrobials, this law is the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Therefore, any claims of sanitizing or disinfecting ductwork would require the use of a product in a manner inconsistent with its labeling, which is a violation of FIFRA. Violations of FIFRA can result in fines and criminal penalties from the EPA.[387]

Another important note about remediation is the myth that you need to "kill the mold." Although many products can easily kill mold, dead mold (if not removed) can be as dangerous as growing mold---due to the continued presence of highly toxic mycotoxins and viable spores which are often impervious to the effects of the agents used to kill the molds.

As the U.S. EPA says:

> The purpose of mold remediation is to remove the mold to prevent human exposure and damage to building materials and furnishings. It is necessary to clean up mold contamination, not just to kill the mold. Dead mold is still allergenic, and some dead molds are potentially toxic.[349]
>
> Biocides are toxic to humans. Do not use fungicides developed for use outdoors for mold remediation or for any other indoor situation.[349]

If someone tells you they have a product that can kill mold or "cure" a mold problem in your home, ask for their peer-reviewed, published research paper that validates the safety and efficacy of their product. To date, none of them have been able to provide such proof.

Cross Contamination

Cross contamination is an important concept because people who have been exposed to toxic mold can cross-contaminate their homes.[391] This can occur if you take contaminated personal items from one house to another or if you carry the contaminants on your hair and body from contaminated buildings. A key issue that is often overlooked in these situations is the nano particulates. These contain 1, 3-Beta-D-glucans, fungal antigens and mycotoxins.[52-53,65-66] They can usually be found during tests of the HVAC system and the ventilation ducts.

> In the case of exposures to mycotoxins including ochratoxin, it is imperative to address issues of cross contamination of items exposed to water-damaged/mold-contaminated environment. Mycotoxins are very difficult to destroy and travel readily on fine, often submicron-sized particles making simple spore testing inadequate for determining the presence of mycotoxins. Thus, a thorough approach is needed to address contamination of items exposed to water-damaged environments to avoid continued exposure to mycotoxins including ochratoxin through these items even if the building is no longer a source of exposure.[278]

Due to the size and weight of fungi particulates, air currents and vibrations, these contaminants are spread quite easily throughout the structure.[52-53,57-59,72,184-186,297,353-357] In addition to attaching itself to clothing, hair and skin (from normal daily activities), the fungal matter can be transported onto every surface. This includes furniture, electronics, clothing and other household material possessions. Computers especially get contaminated as their cooling fans pull in the spores and toxins. Vacuuming without a HEPA filter also spreads the contaminants, because the spores are spread through the air.

Another source of cross contamination occurs when mold remediation is done incorrectly. Quite often, industry guidelines for mold remediation are <u>not</u> followed. In addition, some companies claim to be mold remediators, but they do not have the training or certifications in this field.

Another source of cross contamination occurs when mold remediation is done incorrectly. Quite often, industry guidelines for mold remediation are not followed. In addition, some companies claim to be mold remediators, but they do not have the training or certifications in this field. And, they are typically not knowledgeable about and do not follow industry guidelines for remediation. As a result, they will spread the contamination by blowing the contaminants throughout the structure and the HVAC system by using fans and not following the proper containment procedures.[52-53,62]

There are significant health risks at play when patients and their personal effects are subjected to contamination in damp, rotting, moldy—whether visible to the eye or hidden in the wall cavities—conditions indoors. Another important aspect of cross contamination involves the

method of transporting personal items that can be remediated and the containers used for those items. Information about this part of the process, as well as how to handle books, photos and memorabilia, can be found in industry guidelines.

There are differing opinions on whether personal items such as books, photos, documents, etc. can be effectively remediated. The general consensus is that all water-damaged, porous items should be discarded. However, some guidelines suggest that photos and papers can be photocopied or scanned and saved to digital media (and originals should then be properly discarded). As a reminder, occupants and workers should use proper containment and personal protective equipment when handling any moldy or water-damaged materials.

Steps must be taken to prevent further exposure to the occupants and workers and to avoid contaminating the vehicles used during transport of these contaminated items (e.g., sealing those items in heavy-duty plastic bags before moving them). The outside of the bags should be cleaned with a damp cloth and a soap or detergent solution or HEPA-vacuumed in the work area (or clean changing room) prior to their transport.[349,358]

If important/original documents must be saved, some individuals have used ozone treatment and then stored those items in a sealed container. However, there is no research that proves that ozone treatments prove the efficacy of this approach.

The combination of an infectious threat (the spores) and poisons riding into the occupants' lungs on the surface of respirable particulates coated with a variety of some of the most toxic substances (mycotoxins) known to humankind represents one of the most serious threats to our public's health and to the health of the individual occupants.

When patients find themselves ill after spending time in highly toxic, damp indoor environments, restoring their health depends on their removal from conditions of continued exposure—in addition to the implementation of appropriate treatment protocols.

They should be evacuated from the contaminated space and separated from their personal effects including, but not limited to, clothing, bedding, furniture, books, papers, computers and other electronic devices—most have fans and all have electrostatic and magnetic fields that attract toxic respirable particulates and spores—as all of these items are vectors for cross contaminating other indoor environments into which they are brought.

Causation

Causation is the final issue to address. Differing levels of proof are required for different audiences, and laws vary from state to state. For example, the Michigan Court of Appeals including the following wording in their opinion:

Plaintiffs do not have to present an expert witness to prove they had suffered physical ailments as a result of exposure to mold. According to the court, it was enough that mold was present and that the plaintiffs had physical ailments that could be attributed to mold exposure. The court left it to the defendant to disprove that the mold exposure did not cause the alleged illnesses.[399]

Proving causation is an issue that has been involved in many major health issues. As one example, we will use the history of silicone gel breast implants. Some women received silicone gel breast implants and years later had ruptures or slow leakages. Many of these women subsequently developed tremendous symptomatology similar to that described in this paper. One report indicated that 97% of women had significant improvement in their auto-immune symptoms by simply removing the failed implant.[400] This level of evidence would be convincing for most persons, including most judges and jury members, however the scientific community demands an even higher level.

Large, Institutional Review Board (IRB) approved, controlled, prospective, double-blinded and reproducible trials are considered the gold standard. Yet, it is very unlikely an IRB will ever approve a prospective study that intentionally exposes humans to aflatoxin, endotoxin, digestive enzymes, polysaccharides, lipopolysaccharides (LPS) or any of the other biological toxins found in WDB to further prove that they cause illness in exposed humans. Regardless, sufficient data are already present in the published literature.[192]

There are numerous studies and research reports regarding the health effects of ingested mycotoxins on animals and humans. In addition, there have also been studies regarding the inhalational effects of mycotoxins on mice, rats, guinea pigs and swine (pigs). However, the naysayers refuse to acknowledge those studies. Because some of these toxins are believed to have been used in the past for biological warfare (and could be used in the future), most of the military studies on these toxins are not available to the public. Guidance on the medical management of biological casualties (including trichothecene mycotoxins) is available from the U.S. military.[401-402]

Here are comments from a few of the inhalation studies that are publicly available:

- A 1987 research study on mice concluded that "inhalation of T-2 mycotoxin is at least 10 times more toxic than systemic administration and at least 20 times more toxic than dermal administration."[403]

- A 1990 research study on the inhalation toxicity of T-2 mycotoxins in the rat and guinea pig.

  In this study, concentration-response parameters were determined for rats and guinea pigs systematically exposed to an aerosol of T-2 toxin. These data show that inhaled T-2 toxin is approximately 20 times more toxic to the rat (0.05 mg T-2/kg body wt inhaled vs 1.0 mg T-2/kg body wt ip) and at least twice as toxic to the guinea pig (0.4 mg T-2/kg body wt inhaled vs 1-2 mg T-2/kg body wt ip) than ip administered T-2 toxin.[404]

- A 3-year research study (1982-1985) funded by the U.S. Army on the inhalation effects of mycotoxins on swine and rats. There were several serious health effects including cardiovascular shock; leukocytosis; myocardial, brain, renal, splenic and pancreatic blood flow decreased; heart and pancreatic lesions; subendocardial hemorrhages; pancreatic edema; microscopic and ultrastructural changes in the heart included myofiber degeneration, vacuolization, necrosis and mineralization with formation of hypercontraction bands, pancreatic changes consisting of acinar degeneration and necrosis which progressed to a diffuse suppurative necrotizing pancreatitis; lesions throughout the heart, mitochondrial swelling, etc.[10]

  Swine and rats were used to study toxic effects of T-2 toxin, diacetoxyscirpenol (DAS), and deoxynivalenol (DON), common trichothecene fungal toxins. According to a previous study, it was estimated that the pigs retained approximately 1/3 of the amount of nebulized toxin. Acute toxicosis from T-2 and DAS is a cardiovascular shock syndrome similar to, but distinct from, that of an endotoxin. The syndrome is similar following exposure by oral, inhalation, or intravascular routes.[10]

- A 1987 study on the inhalation effects of T-2 toxin (Trichothecene mycotoxin) on swine.

  Clinically, all of the T-2 treated pigs vomited and were cyanotic, anorexic, lethargic and laterally recumbent. In the 0.33-, 1-, and 3-day T-2 treated pigs, there was a marked reduction in AM phagocytosis and mitogen-induced blastogenic responses of PL but not of peripheral blood lymphocytes. Mild to moderate, multifocal interstitial pneumonia was seen in the majority of the T-2 treated pigs. In pigs dying following inhalation of T-2 toxin, there was a more severe pneumonia, as well as marked necrosis of lymphoid tissues, severe necrohemorrhagic gastroenteritis and edema of the gall bladder wall, and multifocal necrosis of the heart and pancreas. Thus, inhalation exposure to T-2 toxin can result in clinical signs and morphologic changes resembling those reported previously in pigs given T-2 toxin intravascularly (iv) at a dose of 1.2 mg/kg (approximate LD50) or greater, as well as death.[405]

- A 2003 study on the inhalation effects on mice of *Streptomyces californicus*:

  The exposure provoked a dose-dependent inflammatory cell response, as detected by the intense recruitment of neutrophils, but the numbers of macrophages and lymphocytes in the airways also increased. The cellular responses corresponded to the dose-dependent increases in inflammation- and cytotoxicity-associated biochemical markers (i.e., levels of albumin, total protein, and lactate dehydrogenase) in bronchoalveolar lavage fluid. The spore exposure increased the number of both activated and nonactivated T lymphocytes.

  These results indicate that the spores of S. californicus are capable of provoking both immunostimulation in lungs (inflammation) and systemic immunotoxicity, especially in the spleen. Thus, S. californicus must be considered a microbial species with potential to cause systemic adverse health effects in occupants of moisture-damaged buildings.[129]

- A 2008 study on the inhalation effects on mice of *Stachybotrys chartarum:*

  Spores of S. chartarum were injected into the trachea of mice from 6 to 18 times over 4-12 weeks, and the lungs were examined by histopathology, morphometrics and haemodynamics. When 1 x 10(4) spores/mouse were injected, histopathological examination showed the development of pulmonary arterial hypertension (PAH). Symmetrical thickening of the intima and media of the pulmonary arterial walls was seen after six injections over 4 weeks. Right ventricular hypertrophy was also evident after 12 injections. PAH was confirmed by the elevation of right ventricular systolic pressure.[406]

- A 2010 report on the inhalation effects on mice of macrocyclic trichothecene mycotoxins:

  These results suggest that nasal inflammation, mucus hypersecretion, and olfactory neurotoxicity could be important adverse health effects associated with short-term, repeated, airborne exposures to macrocyclic trichothecenes.[135]

- A 2016 report by Dr. Harriett Ammann:

  Inhalation exposure gives direct access to the general circulation through the alveoli, without a first pass through the liver for detoxification as the ingestion route does. Inhalation exposure also provides a pathway to the central nervous system along the olfactory and trigeminal nerve axons in the nasal sensory epithelium that bypasses the blood–brain barrier. The brain is generally shielded from contaminants or drugs through the action of the blood–brain barrier.

  Secondary metabolites of microfungi (molds) and bacteria are present on and in spores and cellular fragments and on dust on which the organisms grow and excrete their toxins, for which the small particle fraction represents the primary exposure medium via inhalation. Deposition of these small particles occurs throughout the respiratory tract, but especially in the alveoli where transport to the bloodstream largely occurs, resulting in toxin distribution to other systemic target organs.[14]

- A 2017 report from Dr. Kakde:

  Exposure to mycotoxins is mostly by ingestion, but also may occur through dermal and inhalation routes (fungal bioaerosols). Some fungal species produce mycotoxins in their spores or in mycelium. The diseases caused by exposure to mycotoxins are known as mycotoxicoses. However, mycotoxicoses often remain unrecognized by medical professionals. Mycotoxins such as Aflatoxin B1 (AFB1), fumonisin B1 (FB1) & ochratoxin A (OTA) which are toxic to mammals causing one of the most toxic effects on them leading to hepatotoxicity, mutagenicity, tetragenicity resulting in diseases like hepatitis, edema, hemorrhage, esophageal cancer & kidney failure. The presence of fungal propagules, volatiles and mycotoxins in the air can cause a health hazard in all segments of the population.[407]

As pointed out by Dr. Shoemaker and his associates, the 2008 US. Government Accountability Office (GAO) report addresses the issue of causation by offering three criteria. If all three criteria are met, then causation has been credibly established for this illness.[408] The three criteria are:

1. epidemiologic associations,

2. experimental exposure in animals or humans that leads to the symptoms and signs of the disease in question, and

3. reduction in exposure that leads to reduction in the symptoms and signs of disease.[10]

In the case of this illness, these criteria have clearly been met, as follows:

1) There are a plethora of studies demonstrating epidemiologic associations between exposure to the interior of WDB (with the associated toxins) and the various symptoms and lab/imaging/neurobehavioral testing found in patients suffering from this illness. Literally tens of thousands of human patients[17-18,192] are also documented in the literature.

2) Many prospective animal studies have been performed which reveal that exposure to various mycotoxins, endotoxins and VOCs have harmful health effects. Re-exposure studies by Dr. Shoemaker *et al* further demonstrate directly that exposure changes symptom scores and lab findings in previously treated humans. In fact, it can be shown reproducibly that patients improve on treatment out of exposure and get worse without treatment when re-exposed.

3) The same re-exposure studies also prove that humans removed from exposure do indeed improve.[17-18,192]

For some individual patients, it is very difficult or impossible to demonstrate that they themselves improve with reduction of exposure because they are unable to limit their presence to certain exposures. An example would be a person made sick in the workplace.

In an ideal world, the patient would take a month or so off work with pay while diagnostic and therapeutic efforts are underway and significant improvement is achieved. Re-exposure upon return to the water-damaged workspace with exacerbation of previous symptoms and lab work would essentially seal the deal. However, many patients will have already exhausted sick leave and vacation time because of previous symptomatology. They are unable financially to remove themselves from the workplace exposure adequately to optimally restore health. These patients will sometimes seek other employment but usually continue working in the environment that is making them ill.

Of course, there can be other factors that can affect an employee's decision to leave or return to the work environment such as the age of the employee, the need to continue working in order to keep their health insurance, the potential for losing their pension or retirement plan, or the possible loss of seniority or tenure (such as in academia).

A few employers are sympathetic and offer testing, remediation and/or accommodations to relieve the problem. This scenario potentially allows the opportunity to prove that the exposure truly is reduced by re-testing after the employer's intervention is implemented with the patient's physician documenting changes in health status. However, many employers do not believe that mold can cause illness and others may feel threatened by potential lawsuits and the hassles of workers' compensation insurance or disability claims. Some will be more concerned by the potential cost of appropriate remediation than the costs to the employee's health.

In these scenarios, where the patient continues to go to work or school in an unmodified space, causation can still be inferred by treating with a sequestering agent such as cholestyramine, charcoal and/or clay. Since their actions are to bind and remove bile acids and the ionically charged molecules attached to them (through the enterohepatic circulation), sequestering agents are intended to reduce total body loads of many substances including many toxins.

Reduced body toxin loads can be monitored via VCS and other targeted diagnostic testing modalities including urine mycotoxin studies (available through RealTime Laboratories in Dallas, Texas).[37-38,266,289,328,338-339] Documented symptoms, reduced by severity, frequency and/or duration, reveal a positive patient response. Resolution of abnormal blood tests further displays disease improvement.

Of note, a common scenario for the persistence of symptoms, after escaping exposure from a water-damaged building, is caused by cross contamination, or the transport of materials which have been contaminated with mycotoxins and other toxic and microbial agents, from the previous water-damaged building to the new setting. This phenomenon results in an ongoing exposure, as there was never any practiced avoidance. A detailed discussion on cross contamination is provided earlier in this paper.

A common scenario for the persistence of symptoms, after escaping exposure from a water-damaged building, is caused by cross contamination, or the transport of materials which have been contaminated with mycotoxins and other toxic and microbial agents, from the previous water-damaged building to the new setting.

Dr. Shoemaker's group has developed and published a re-exposure trial known as the ABB´AB protocol.[17-18,35] The first "A" stands for a patient who is ill in an exposure.

The first "B" represents the same patient—now improved on therapy and out of exposure. B´ refers to an improved patient who remains out of exposure and maintains improved health off of therapy.

The second "A" indicates that the patient, off therapy, is re-exposed to the previous suspected environment for at least three days.

The final "B" documents the patient once again on therapy after the re-exposure. Symptom logs, VCS testing and specific labs are obtained just prior to re-exposure and at 24, 48 and 72 hours after re-exposure. If the suspected environment is an exposure, and hence a health risk for this patient, symptom scores will increase, VCS scores will decrease and certain lab tests will increase and/or decrease predictably in post-exposure levels compared to those obtained just prior to re-exposure.[17-18,23,33,35,158,,192-193,236]

While performing the ABB´AB protocol may not be required to establish causation for a patient, successful administration of this trial is very powerful evidence, difficult to refute, and clearly meets all three GAO-suggested criteria for causation between a purported exposure and an individual. It is a prospective re-exposure trial with the only variable being re-introduction to the suspected sick building.[17-18,33,192]

It is also helpful to test one or more suspected environments to document the presence of water damage. A water-damaged building, by definition, will have multiple species of molds, bacteria, etc. There are a number of testing strategies and using a combination of methods is recommended by many. A detailed discussion about testing is provided earlier in this paper.

Verifying the presence of water damage in the interior of a building proves the existence of potentially harmful microbial agents and the secondary metabolites they exude. As noted above, it is not possible, nor necessary, to identify the individual toxin(s) which is (are) harming the human host. That there is at least one toxin in the interior of a WDB causing disease is inferred by the presence of water damage (causation has already been established as above) and the patient's improvement on a sequestration protocol with/without removal from the exposure.

In the words of the WHO 2009 report (Executive Summary, p. XV):

> As the relations between dampness, microbial exposure and health effects cannot be quantified precisely, no quantitative health-based guideline values or thresholds can be recommended for acceptable levels of contamination with microorganisms. Instead, it is recommended that dampness and mould-related problems be prevented.[1]

Examples of causation during litigation

There have been several important court rulings and settlements in favor of plaintiffs in regard to causation. The outcomes of four important cases are cited here:

From a New York Supreme Court ruling (Granirer vs The Baker, Inc., et al)[409]:

Plaintiffs' evidence provides a scientific expression' of plaintiffs' exposure levels to several species of toxigenic fungi — that is, fungi that produce mycotoxins — in quantities evidencing high levels of contamination. Thus, plaintiffs have laid an adequate foundation for their opinions on specific causation.

From a Michigan Court of Appeals ruling (Genna vs Jackson, et al)[399]:

Plaintiffs do not have to present an expert witness to prove they had suffered physical ailments as a result of exposure to mold. According to the court, it was enough that mold was present and that the plaintiffs had physical ailments that could be attributed to mold exposure. The court left it to the defendant to disprove that the mold exposure did not cause the alleged illnesses.

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From an article about a $22 Million Settlement in a mold lawsuit (Gorman vs Crenshaw Lumber, et al)[410]:

As the plaintiff's attorney stated, "This is a case that puts attention on the fact that mold exposure can lead not just to the old culprits of respiratory pulmonary problems but can lead to brain injuries through an immunologic process."

From an article about a $11.78 Million Settlement in a mold lawsuit (Gage vs Philadelphia Housing Authority, et al)[411]:

The case was settled just before opening statements of the trial. As part of the settlement, the District Court agreed to vacate its April 20 summary judgment ruling.

When treating patients only (i.e., litigation is not involved), it is not necessary to prove that a school or place of employment is the only exposure implicated. The treating practitioner's recommendation will be to test and/or remediate all spaces with water damage to which the patient is exposed. However, those who administer schools and workplaces are mandated by various laws and agencies to provide safe facilities for their students and employees. As such, even if a school or business is not the only WDB to which the patient is exposed, proper testing, remediation as needed and re-testing of these places must be performed.

Landlords have a similar duty, although it is typically difficult to get them to make the necessary repairs or to pay for proper remediation. In some cases, it has been helpful to get assistance from local building code enforcement or public health officials. Other tenants have been able to get help from their elected officials in the state or federal legislature. It may also help to get media involved.

The practitioner should always keep in mind, however, the possibility of future (or current) litigation. As such, documentation is the most basic key to demonstrating causation. Documenting worsening of the condition with re-exposures is also important.

Conclusion

In summary, illness caused by exposure to contaminants in water-damaged buildings is a multi-symptom, multi-system disease.

While a massive acute exposure can lead to this illness, the most common mechanism is chronic exposure to low level toxin leading to an inflammatory response in the body.

Unfortunately, most physicians will not recognize the illness because they are uninformed about the variable multi-system presentations or have been misinformed about the serious health risks of molds, mycotoxins and other contaminants inside water-damaged buildings.

The tide is turning. Knowledge and awareness of this illness is spreading, and the diagnosis and treatment protocols are being shared around the world.

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Global Indoor Health Network

The Global Indoor Health Network (GIHN) is a 501(c)(3) nonprofit organization dedicated to providing education and awareness of the health effects of mold and other indoor contaminants. We are uniting experts and laypersons from the world, with members throughout the United States and in eleven other countries. GIHN's vision is a global community of individuals and organizations working together to ensure that comprehensive information and guidance concerning medical treatment, investigative techniques and solutions are available to address the effects of contaminants in the indoor environment of homes, schools and businesses.

Visit our website at: https://www.globalindoorhealthnetwork.com.

References

1. World Health Organization (WHO). WHO Guidelines for Indoor Air Quality – Dampness and Mould. http://www.who.int/indoorair/publications/7989289041683/en/. Published 2009.
2. Ammann HM. Indoor Mold Contamination--a Threat to Health? *J Environ Health*. 2002;64(6):43.
3. Ammann HM. Indoor Mold Contamination--a Threat to Health? Part Two. *J Environ Health*. 2003 Sep 1;66(2):47.
4. Daschner A. An Evolutionary-Based Framework for Analyzing Mold and Dampness-Associated Symptoms in DMHS. *Front Immunol.* January 2017;7(672). https://doi.org/10.3389/fimmu.2016.00672.
5. McMahon SW, Hope JH, Thrasher JD, Rea WJ, Vinitsky AR, Gray MR. Global Indoor Health Network (GIHN). Common Toxins in Our Homes, Schools and Businesses. December 17, 2012. (This 2012 position statement by GIHN has been replaced with new papers on individual topics.)
6. Gray MR. Progressive Healthcare Group. http://www.phgaz.com.
7. Gray MR. Cumulative Organic Chemical Hyper-Toxicity (COCHT). Educational videos. https://www.youtube.com/user/MyChemicalAllergy.
8. International Society for Environmentally Acquired Illness (ISEAI). https://iseai.org.
9. World Health Organization (WHO), Regional Office for Europe Copenhagen. Indoor Air Pollutants: Exposure and Health Effects. Report on a WHO Meeting. June 8-11, 1982.
10. U.S. Army Medical Research and Development Command, with the College of Veterinary Medicine, University of Illinois. Toxicologic and Analytical Studies with T-2 and Related Trichothecene Mycotoxins (3-year study on the inhalation effects of mycotoxins). August 20, 1985, Contract No. DAMD17-82-C-2179Etzel RA. Toxic Effects of Indoor Molds. *Amer Acad Pediatr*. 1998 Apr;101(4)712-714.
11. Zajtchuk R, Bellamy RF, eds. Medical Aspects of Chemical and Biological Warfare. Textbook of Military Medicine. Office of the Surgeon General, Department of the Army, USA. 1997.
12. Forgacs J, Carll WT. Mycotoxicosis. *Adv Vet Sci*. 1962;7:273-293.
13. Forgacs J. Stachybotryotoxicosis. Microbial Toxins Vol. III. Published by Academic Press. 1972:95-128.
14. Ammann HM. Inhalation Exposure and Toxic Effects of Mycotoxins. *Biology of Microfungi*. 2016:495-523. https://doi.org/10.1007/978-3-319-29137-6_20.
15. American Conference of Governmental Industrial Hygienists (ACGIH). Bioaerosols: Assessment and Control. Published by ACGIH. 1999.
16. Engvall K, Norrby C, Norbäck D. Sick Building Syndrome in Relation to Building Dampness in Multi-Family Residential Buildings in Stockholm. *Int Arch Occup Environ Health*. 2001 May;74(4):279-278. https://doi.org/10.1007/s004200000218.
17. Shoemaker RC. Mold Warriors: Fighting America's Hidden Health Threat. Published by Gateway Press. April 2005.
18. Shoemaker RC. Surviving Mold: Life in the Era of Dangerous Buildings. Published by Otter Bay Books. December 15, 2010.
19. Johanning E. Bioaerosols, Fungi, Bacteria, Mycotoxins and Human Health. Published by Fungal Research Group Foundation. March 1,2005:508 pages.
20. Johanning E. Bioaerosols, Fungi and Mycotoxins, Health Effects, Assessment, Prevention and Control. Published by Eastern New York Occupational and Environmental Health. December 31, 1999:638 pages.
21. Bennett JW, Klich M. Mycotoxins. *Clin Microbiol Rev*. 2003 Jul;16(3): 497-516. doi:10.1128/CMR.16.3.497-516.2003.
22. Kilburn KH. Towards Healthy Homes. *Toxicol Ind Health*. 2009;25(9-10):737-740. doi:10.1177/0748233709351442.
23. Nathan N. Mold and Mycotoxins: Current Evaluation & Treatment. Published by BookBaby. July 17, 2016.
24. Azuma K, Ikeda K, Kagi N, Yanagi U, Hasegawa K, Osawa H. Effects Of Water-Damaged Homes After Flooding: Health Status of the Residents and the Environmental Risk Factors. *Int J Environ Health Res*. 2014 Apr;24(2):158-175. doi:10.1080/09603123.2013.800964.
25. Billings K, Billings LA. Mold: The War Within. Published by Partners Publishing LLC. June 1, 2013.
26. Croft WA, Jarvis BB, Yatawara CS. Airborne Outbreak of Trichothecene Toxicosis. *Atmos Environ*. 1986;20(3):549–552. https://doi.org/10.1016/0004-6981(86)90096-X.

27. Johanning E, Biagini R, Hull D, Morey P, Jarvis B, Landsbergis P. Health and Immunology Study Following Exposure to Toxigenic Fungi (*Stachybotrys chartarum*) in Water-Damaged Office Environment. *Int Arch Occ Environ Health*. 1986;68(4):207-218. https://doi.org/10.1007/BF00381430.
28. Hodgson MJ, Morey P, Leung WY, et al. Building-Associated Pulmonary Disease from Exposure to *Stachybotrys chartarum* and *Aspergillus versicolor*. *Int J Occ Environ Med*. 1988;40(3):241-249.
29. Gray MR, Thrasher JD, Crago R, et al. Mixed Mold Mycotoxicosis: Immunological Changes in Humans Following Exposure in Water-Damaged Buildings. *Arch Environ Health*. 2003 Jul;58(7):410-420. https://doi.org/10.1080/00039896.2003.11879142.
30. Cooley JD, Wong WC, Jumper CA, Straus DC. Fungi and the Indoor Environment: Their Impact on Human Health. *Adv Appl Microbiol*. 2004;55:1-30. doi:10.1016/S0065-2164(04)55001-3.
31. Rea WJ, Didriksen N, Simon TR, et al. Effects of Toxic Exposure to Molds and Mycotoxins in Building-Related Illnesses. *Arch Environ Health*. 2003;58(758):399–405. https://doi.org/10.1080/00039896.2003.11879140.
32. Etzel, RA. What the Primary Care Pediatrician Should Know about Syndromes Associated with Exposures to Mycotoxins. *Curr Probl Pediatr Adolesc Health Care*. September 2006;36(8):282-305. https://doi.org/10.1016/j.cppeds.2006.05.003.
33. Shoemaker RC, House DE. Sick Building Syndrome (SBS) and Exposure to Water-Damaged Buildings: Time Series Study, Clinical Trial and Mechanisms. *Neurotoxicol Teratol*. 2006;28(5):573-588. https://doi.org/10.1016/j.ntt.2006.07.003.
34. Hope JH. A Review of the Mechanism of Injury and Treatment Approaches for Illness Resulting from Exposure to Water-Damaged Buildings, Mold and Mycotoxins. *The Scientific World Journal*. 2013;article ID 767482:20 pages. 2013. http://dx.doi.org/10.1155/2013/767482.
35. Berndtson, Keith. Mold Toxicity: A Chronic Inflammatory Response Syndrome (presentation). December 17, 2013.
36. Thrasher JD. Gray MR, Kilburn KH, Dennis DP, Yu A. A Water-Damaged Home and Health of Occupants: A Case Study. *J Environ Public Health*. 2012:article ID 312836:10 pages. http://dx.doi.org/10.1155/2012/312836.
37. Thrasher JD, Hooper DH, Taber, J. Family of Six, their Health and the Death of a 16-Month-Old Male from Pulmonary Hemorrhage: Identification of Mycotoxins and Mold in the Home and Lungs, Liver and Brain of Deceased Infant. *Global J Med Res: K Interdisciplinary*. 2014;14(5)Version 1.0.
38. Thrasher JD, Prokop C, Roberts C, Hooper D. A Family with ME/CFS Following Exposure to Molds, Mycotoxins and Bacteria in a Water-Damaged Home: A Case Report. *Int J Clin Toxicol*. 2016;4:14-23.
39. Tuuminen T, Rinne KS. Severe Sequelae to Mold-Related Illness as Demonstrated in Two Finnish Cohorts. *Front Immunol*. 2017 Apr 3;8(382). https://doi.org/10.3389/fimmu.2017.00382.
40. Curtis L, Lieberman A, Stark M, Rea W, Vetter M. Adverse Health Effects of Indoor Molds. *J Nutrit Environ Med*. September 2004;14(3):261-274. https://doi.org/10.1080/13590840400010318.
41. Mudarri D, Fisk WJ. Public Health and Economic Impact of Dampness and Mold. *Indoor Air*. 2007 Jun;17(3):226-235. https://doi.org/10.1111/j.1600-0668.2007.00474.x.
42. Kilburn K. Neurobehavioral and Pulmonary Impairment in 105 Adults with Indoor Exposure to Molds Compared to 100 Exposed to Chemicals. *Toxicol Ind Health*. 2009 Oct-Nov;25(9-10):681-92. https://doi.org/10.1177/0748233709348390.
43. Institute of Medicine of the National Academies (IOM). Beyond Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: Redefining an Illness. *The National Academies Press*. February 2015:330 pages.
44. National Institute of Occupational Safety and Health (NIOSH). NIOSH Alert: Preventing Occupational Respiratory Disease from Exposures Caused by Dampness in Office Buildings, Schools, and Other Nonindustrial Buildings. Publication No. 2013–102. https://www.cdc.gov/niosh/docs/2013-102/pdfs/2013-102.pdf. Published November 2012.
45. Dedesko S, Siegel JA. Moisture Parameters and Fungal Communities Associated with Gypsum Drywall in Buildings. *Microbiome*. 2015;3:71. https://doi.org/10.1186/s40168-015-0137-y.
46. Andersen B, Dosen I, Lewinska AM, Nielsen KF. Pre-Contamination of New Gypsum Wallboard with Potentially Harmful Fungal Species. *Indoor Air:* 2017;27(1):6-12. https://doi.org/10.1111/ina.12298.
47. Gravesen S, Nielsen PA, Iversen R, Nielsen KF. Microfungal Contamination of Damp Buildings – Examples of Risk Constructions and Risk Materials. *Environ Health Perspect*. 1999 Jun;107(Suppl 3):505–508.
48. Michigan State University Extension. Wood-Damaging Fungi: Chapter 7 of Management of Wood-Destroying Pests. December 2000;Extension Bulletin E-2047.

49. Andersen B, Dosen I, Lewinska AM, Nielsen KF. Pre-Contamination of New Gypsum Wallboard with Potentially Harmful Fungal Species. *Indoor Air*. 2017 Jan;27(1):6-12. https://doi.org/10.1111/ina.12298.
50. U.S. Department of Housing and Urban Development (HUD). Healthy Homes Issues: Mold. Version 3. March 2006. https://www.hud.gov/sites/documents/DOC_12483.PDF.
51. U.S. Environmental Protection Agency (EPA). Mold Course, Chapter 2: Why and Where Mold Grows. Page last updated on February 21, 2017. https://www.epa.gov/mold/mold-course-chapter-2.
52. Thrasher JD. The Biocontaminants and Complexity of Damp Indoor Spaces: More Than What Meets the Eyes. *Toxicol Ind Health*. 2009;25(9-10):583-615. https://doi.org/10.1177/0748233709348386.
53. Thrasher JD. Fungi, Bacteria, Nano-Particulates, Mycotoxins and Human Health in Water-Damaged Indoor Environments. *Journal of Community and Public Health Nursing*. 2016;2(2):8 pages. http://dx.doi.org/10.4172/jcphn.1000115.
54. Lee TG. Health Symptoms Caused by Molds in a Courthouse. Published in Molds and Mycotoxins. *Arch Environ Health*. Dr. Kaye H. Kilburn, M.D. (ed.) Heldref Publications. 2003;58(7):442-446. https://doi.org/10.1080/00039896.2003.11879145.
55. Occupational Safety and Health Organization (OSHA). Indoor Air Quality in Commercial and Institutional Buildings. 2011;3430-04:28 pages. https://www.osha.gov/Publications/3430indoor-air-quality-sm.pdf.
56. Yang CS. Biological Contamination in the HVAC System. P&K Microbiology Services, Inc. 2000. *INvironment*. http://www.abatement.com/media/pdf/biological-contaminants-hvac-system.pdf.
57. Rao CY, Riggs MA, Chew GL, et al. Characterization of Airborne Molds, Endotoxins, and Glucans in Homes in New Orleans after Hurricanes Katrina and Rita. *Appl Environ Microbiol*. 2007;73(5):1630-1634. doi:10.1128/AEM.01973-06.
58. Rylander R. Airborne (1→3)-β-d-Glucan Disease in a Day-Care Center Before and After Renovation. *Arch Environ Health*. 1997;52:281-285. https://doi.org/10.1080/00039899709602199.
59. Rylander R, Norhall M, Engdahl U, Tunsater A, Holt PG. Airway Inflammation, Atopy and (1→3)-β-d-Glucan Exposure in Two Schools. *Amer J Respir Crit Care Med*. 1998;158(5 Pt 1):1685-1687. https://doi.org/10.1164/ajrccm.158.5.9712139.
60. Chew GL, Douwes J, Doekes G, Higgins KM, van Strien R, Spithoven J, Brunekreef B. Fungal Extracellular Polysaccharides, β (1→3)-Glucans and Culturable Fungi in Repeated Sampling of House Dust. *Indoor Air.* 2001;11(3):171-178. https://doi.org/10.1034/j.1600-0668.2001.011003171.x.
61. Gehring U, Douwes J, Doekes G, et al. Beta(1-->3)-glucan in House Dust of German Homes: Housing Characteristics, Occupant Behavior, and Relations with Endotoxins, Allergens, and Molds. *Environ Health Perspect*. 2001;109(2):139-144.
62. Beijer L, Thorn T, Rylander R. Effects After Inhalation of (1 → 3)-β-D-glucan and Relation to Mould Exposure in the Home. *Mediators Inflamm*. 2002;11(3):149-53. http://dx.doi.org/10.1080/09622935020138181.
63. Douwes J, Siebers R, Wouters I, Doekes G, Fitzharris P, Crane J. Endotoxin, (1 --> 3)-beta-D-glucans and Fungal Extra-Cellular Polysaccharides in New Zealand Homes: A Pilot Study. *Ann Agric Environ Med*. 2006;13(2):361-365.
64. Giovannangelo ME, Gehring U, Nordling E, et al. Levels and Determinants of (1-3)-β-glucans and Fungal Extracellular Polysaccharides in House Dust of (Pre-) School Children in Three European Countries. *Environ Int*. 2007;33(1):9-16. https://doi.org/10.1016/j.envint.2006.06.018.
65. Seo S-C, Reponen T, Levin L, Borchdelt T, Grinshpun SA. Aerosolization of Particulate (1→3)-β-D-Glucan from Moldy Materials. *Appl Environ Microbiol*. 2008;74(3):585-593.
66. Seo S-C, Reponen T, Levin L, Grinshpun SA. Size-Fractionated (1→3)-β-D-Glucan Concentrations Aerosolized from Different Moldy Building Materials. *Sci Total Environ*. 2009;407(2):806-814. https://doi.org/10.1016/j.scitotenv.2008.10.018.
67. Charpin-Kadouch C, Maurel G, Felipo R, et al. Mycotoxin Identification in Moldy Dwellings. *J Appl Toxicol*. 2006;26(6):475-479. https://doi.org/10.1002/jat.1164.
68. Madsen AM, Larsen ST, Koponen IK, et al. Generation and Characterization of Indoor Fungal Aerosols for Inhalation Studies. *Appl Environ Microbiol*. 2016 Apr;82(8):2479-2493. doi:10.1128/AEM.04063-15.
69. U.S. Centers for Disease Control and Prevention (CDC). Mold Prevention Strategies and Possible Health Effects in the Aftermath of Hurricanes and Major Floods. MMWR (Morbidity and Mortality Weekly Report) Recommendations and Reports. June 9, 2006;55(RR08):1-27.

70. Choi H, Schmidbauer N, Bornehag CG. Volatile Organic Compounds of Possible Microbial Origin and Their Risks on Childhood Asthma and Allergies within Damp Homes. *Environ Int*. 2017 Jan;98:143-151. https://doi.org/10.1016/j.envint.2016.10.028.
71. Inamdar AA, Hossain MM, Bernstein AI, Miller GW, Richardson JR, Bennett JW. Fungal-derived Semiochemical 1-octen-3-ol Disrupts Dopamine Packaging and Causes Neurodegeneration. Proceedings of the National Academy of Sciences. 2013 Nov 26;110(48):19561-19566. https://doi.org/10.1073/pnas.1318830110.
72. Betancourt DA, Krebs K, Moore SA, Martin SM. Microbial Volatile Organic Compound Emissions from *Stachybotrys chartarum* Growing on Gypsum Wallboard and Ceiling Tile. *BMC Microbiology*. 2013;13:283. https://doi.org/10.1186/1471-2180-13-283.
73. Inamdar AA, Bennett JW. A Common Fungal Volatile Organic Compound Induces a Nitric Oxide Mediated Inflammatory Response in *Drosophila melanogaster*. *Sci Rep*. 2014 Feb 10;4:article 3833. doi:10.1038/srep03833.
74. Hung R, Lee S, Bennett JW. Fungal Volatile Organic Compounds and Their Role in Ecosystems. *Appl Microbiol Biotechnol*. 2015 Apr;99(8):3395-3405. https://doi.org/10.1007/s00253-015-6494-4.
75. Zhao G, Yin G, Inamdar AA, et al. Volatile Organic Compounds Emitted by Filamentous Fungi Isolated from Flooded Homes After Hurricane Sandy Show Toxicity in a *Drosophila* Bioassay. *Indoor Air*. 2017 May;27(3):518-528. https://doi.org/10.1111/ina.12350.
76. Lemfack MC, Nickel J, Dunkel M, Preissner R, Peichulla B. mVOC: A Database of Microbial Volatiles. *Nucleic Acids Res*. 1 January 2014;42(D1):D744–D748. https://doi.org/10.1093/nar/gkt1250.
77. Pessi AM, Suonketo J, Pentti M, Kurkilahti M, Peltola K, Rantio-Lehtimaki A. Microbial Growth Inside Insulated External Walls as an Indoor Air Biocontamination Source. *Appl Environ Microbiol*. 2002;68(2):963-967. doi:10.1128/AEM.68.2.963-967.2002.
78. Hope AP, Simon RA. Excess Dampness and Mold Growth in Homes: An Evidence-Based Review of the Aero-Irritant Effect and its Potential Causes. *Allergy Asthma Proc*. 2007;28(3):262-270(9). https://doi.org/10.2500/aap.2007.28.3004.
79. Menzies D, Comtois P, Pasztor J, Nunes F, Hanley JA. Aeroallergens and Work-Related Respiratory Symptoms among Office Workers. *J Allergy Clin Immunol*. 1998;101(1):38-44. https://doi.org/10.1016/S0091-6749(98)70191-5.
80. Bornehag CG, Sundell J, Sigsgaard T. Dampness in Buildings and Health (DBH): Report from an Ongoing Epidemiological Investigation on the Association between Indoor Environmental Factors and Health Effects among Children in Sweden. *Indoor Air.* 2004;14(Suppl 7):59-66.
81. Marinkovich VA. Fungal Hypersensitivity: Pathophysiology, Diagnosis, Therapy. *Adv Appl Microbiol*. 2004;55:289-307.
82. Park JH, Schleiff PL, Attfield MD, Cox-Ganser JM, Kreiss K. Building-Related Respiratory Symptoms Can be Predicted with Semi-Quantitative Indices of Exposure to Dampness and Mold. *Indoor Air*. 2004;14:425-433. https://doi.org/10.1111/j.1600-0668.2004.00291.x.
83. Cho SH, Seo SC, Schmechel D, Grinshpun SA, Reponen A. Aerodynamic Characteristics and Respiratory Deposition of Fungal Fragments. *Atmos Environ*. 2005;39(30):5454-5465. https://doi.org/10.1016/j.atmosenv.2005.05.042.
84. Dangman KH, Bracker AL, Storey E. Work-Related Asthma in Teachers in Connecticut Schools with Chronic Water Damage and Fungal Growth. *Conn Med*. 2005;69(1):9-17.
85. Cox-Ganser JM, White SK, Jones R, et al. Respiratory Morbidity in Office Workers in a Water-Damaged Building. *Environ Health Perspec*. 2005 Apr;113(4):485-90. doi:10.1289/ehp.7559.
86. Jaakkola JJK, Hwang BF, Jaakkola N. Home Dampness and Molds, Parental Atopy, and Asthma in Childhood: A Six-Year Population-Based Cohort Study. *Environ Health Perspect*. 2005;113(3):357–361. doi:10.1289/ehp.7242.
87. Denning DW, O'Driscoll BR, Hogaboam CM, Bowyer P, Niven RM. The Link Between Fungi and Severe Asthma: A Summary of the Evidence. *Eur Respir J*. 2006;27:615-626. doi:10.1183/09031936.06.00074705.
88. Jaakkola JJK, Ieromnimon A, Jaakkola MS. Interior Surface Materials and Asthma in Adults: A Population-Based Incident Case-Control Study. *Am J Epidemiol*. October 15, 2006;164(8):742–749. https://doi.org/10.1093/aje/kwj249.

89. Park J-H, Cox-Ganser JM, Kreiss K, White SK, Rao CV. Hydrophilic Fungi and Ergosterol Associated with Respiratory Illness in a Water-Damaged Building. *Environ Health Perspect*. 2008 Jan;116:45-50. doi:10.1289/ehp.10355.
90. Laney AS, Cragin LA, Blevins LZ, et al. Sarcoidosis, Asthma and Asthma-Like Symptoms Among Occupants of a Historically Water-Damaged Office Building. *Indoor Air*. February 2009;19:83-90. https://doi.org/10.1111/j.1600-0668.2008.00564.x.
91. Fisk WJ, Eliseeva EA, Mendell MJ. Association of Residential Dampness and Mold with Respiratory Tract Infections and Bronchitis: A Meta-Analysis. *Environ Health*. 2010;9:72:1-11 pages. https://doi.org/10.1186/1476-069X-9-72.
92. Karvala K, Toskala E, Luukkonen R, Lappalainen S, Uitti J, Nordman H. New-Onset Adult Asthma in Relation to Damp and Moldy Workplaces. *Int Arch Occ Environ Health*. 2001;83(8):855–865. https://doi.org/10.1007/s00420-010-0507-5.
93. Mendell MJ, Mirer AG, Cheung K, et al. Respiratory and Allergic Health Effects of Dampness, Mold and Dampness-Related Agents: A Review of Epidemiologic Evidence. *Environ Health Perspect*. 2011 Jun;119(6):748-756. doi:10.1289/ehp.1002410.
94. Park J-H, Cox-Ganser JM. Mold Exposure and Respiratory Health in Damp Indoor Environments. *Frontiers Biosci*. 2011;E3:757-71. doi:10.2741/e284.
95. Reponen T, Vesper S, Levin L, et al. High Environmental Relative Moldiness Index During Infancy as a Predictor of Asthma at 7 Years of Age. *Ann Allergy Asthma Immunol*. 2011;107(2):120-126. https://doi.org/10.1016/j.anai.2011.04.018.
96. Croston TL, Lemons AR, Beezhold DH, Green BJ. MicroRNA Regulation of Host Immune Responses following Fungal Exposure. *Front. Immunol*. 2018 Feb 07;9(170)1-11. https://doi.org/10.3389/fimmu.2018.00170.
97. Croston TL, Nayak AP, Lemons AR, et al. Pulmonary Immune Response Following Subchronic *Stachybotrys chartarum* Exposure. *J Allergy Clin Immunol*. 2017;139(2):AB75. doi:10.1016/j.jaci.2016.12.291.
98. Etzel RA, Montaña E, Sorenson, WG, et al. Acute Pulmonary Hemorrhage in Infants Associated with Exposure to *Stachybotrys atra* and Other Fungi. *Arch Pediatr Adolesc Med*. 1998;152(8):757-762. doi:10.1001/archpedi.152.8.757.
99. Apostolakos MJ, Rossmoore H, Beckett WS. Hypersensitivity Pneumonitis from Ordinary Residential Exposures. *Environ Health Perspect*. 2001 Sep;109(9):979–981.
100. Rylander R. The Link Between Exposure to Mould and Respiratory Problems is Incontrovertible. *Lakartidningen*. 1998 May 20;95(21):2445-2446.
101. Creasia DA, Nealley ML, Jones LJ, York CG, Wannemacher RW, Bunner DL. Acute Inhalation Toxicity of a Saline Suspension of T-2 Mycotoxin in Mice. U.S. Army Medical Research Inst of Infectious Disease. *Toxicol Appl Pharmacol*. 1986 Nov 07. http://www.dtic.mil/dtic/tr/fulltext/u2/a190156.pdf.
102. Park J-H, Cho SJ, White SK, Cox-Ganser JM. Changes in Respiratory and Non-Respiratory Symptoms in Occupants of a Large Office Building over a Period of Moisture Damage Remediation Attempts. *PLoS ONE*. 2018;13(1):e0191165. https://doi.org/10.1371/journal.pone.0191165.
103. Dickson SD, Tankersley MS. Fatal Hypersensitivity Pneumonitis from Exposure to *Fusarium vasinfectum* in a Home Environment: A Case Report. *Int Arch Allergy Immunol*. 2015;166(2):150-153. doi:10.1159/000377631.
104. Park J-H, Kreiss K, Cox-Ganser JM. Rhinosinusitis and Mold as Risk Factors for Asthma Symptoms in Occupants of a Water-Damaged Building. *Indoor Air*. 2012;22(5):396-404. doi:10.1111/j.1600-0668.2012.00775.x.
105. Chen CH, Chao HJ, Chang-Chuan CC, Chen BY, Guo YL. Current Asthma in Schoolchildren is Related to Fungal Spores in Classrooms. *Chest*. 2014;146(1):123-134. doi:10.1378/chest.13-2129.
106. Bornehag CG, Blomquist G, Gyntelberg F, et al. Dampness in Buildings and Health. Nordic Interdisciplinary Review of the Scientific Evidence on Associations Between Exposure to "Dampness" in Buildings and Health Effects (NORDDAMP). *Indoor Air*. 2001 Jun;11(2):72-86.
107. Campbell AW, Thrasher JD, Madison RA, et al. Neural Antibodies and Neurophysiologic Abnormalities in Patients Exposed to Molds in Water-Damaged Buildings. *Arch Environ Health*. August 2003;58(8):464-74. https://doi.org/10.3200/AEOH.58.8.464-474.
108. LaDou J, Teitelbaum DT, Egilman DS, Frank AL, Kramer SN, Huff J. American College of Occupational and Environmental Medicine (ACOEM): A Professional Association in Service to Industry. Int J Occup Environ Health. 2007 Oct/Dec;13(4):404-426.
109. Armstrong D. Amid Suits Over Mold, Experts Wear Two Hats. The Wall Street Journal. January 9, 2007.

110. Global Indoor Health Network. Discussion of Naysayers and Deniers. September 2017. https://www.globalindoorhealthnetwork.com/GIHN-papers.
111. Anyanwu EC, Campbell AW, Vojdani A. Neurophysiological Effects of Chronic Indoor Environmental Toxic Mold Exposure on Children. *The Scientific World Journal*. 2003;3:281-290. http://dx.doi.org/10.1100/tsw.2003.22.
112. Empting, LD. Neurologic and Neuropsychiatric Syndrome Features of Mold and Mycotoxin Exposure. *Toxicol Ind Health*. 2009;25(9-10):577-581. https://doi.org/10.1177/0748233709348393.
113. Gordon WA, Cantor JB, Johanning E, et al. Cognitive Impairment Associated with Toxigenic Fungal Exposure: A Replication and Extension of Previous Findings. *Appl Neuropsychol*. 2004;11:65-74. https://doi.org/10.1207/s15324826an1102_1.
114. Campbell AW, Thrasher JD, Gray MR, Vojdani A. Mold and Mycotoxins: Effects on the Neurological and Immune Systems in Humans. *Adv Appl Microbiol*. 2004;55:375-406.
115. Anyanwu E, Campbell AW, Jones J, Ehiri JE. The Neurological Significance of Abnormal Natural Killer Cell Activity in Chronic Toxigenic Mold Exposures. *Sci World J*. 2003;3 1128-1137. http://dx.doi.org/10.1100/tsw.2003.98.
116. Lieberman A, Curtis L, Campbell A. Development of New-Onset Chronic Inflammatory Demyelinating Polyneuropathy Following Exposure to a Water-Damaged Home with High Airborne Mold Levels: A Report of Two Cases and a Review of the Literature. *J. Neurol Res*. 2017;7(3):59-62. https://doi.org/10.14740/jnr413e.
117. Baldo JV. Neuropsychological Performance of Patients Following Mold Exposure. *Appl Neuropsychol*. 2002;9(4):193-202. https://doi.org/10.1207/S15324826AN0904_1.
118. Crago, BR, Gray MR, Nelson LA, Davis M, Arnold L, Thrasher JD. Psychological, Neuropsychological, and Electrocortical Effects of Mixed Mold Exposure. *Arch Environ Health*. 2003;58(8)452-463. https://doi.org/10.3200/AEOH.58.8.452-463.
119. Anyanwu EC. The Validity of the Environmental Neurotoxic Effects of Toxigenic Molds and Mycotoxins. *The Internet Journal of Toxicology*. 2008;5(2).
120. Karunasena E, Larranaga MD, Simoni JS, Douglas DR, Straus DC. Building-Associated Neurological Damage Modeled in Human Cells: A Mechanism of Neurotoxic Effects by Exposure to Mycotoxins in the Indoor Environment. *Mycopathologia*. 2010 Dec;170(6):377-390. https://doi.org/10.1007/s11046-010-9330-5.
121. Singer R. B-93 Neuropsychological Development Disorder Following Mold Exposure. *Arch Clin Neuropsych*. 2013;28(6):518-626. doi:10.1093/arclin/act054.188.
122. Abou-Donia MB, Lieberman A, Curtis L. Neural Autoantibodies in Patients with Neurological Symptoms and Histories of Chemical/Mold Exposure. *Toxicol Ind Health*. 2018;34(10).
123. Doi K, Uetsuka K. Mechanisms of Mycotoxin-Induced Neurotoxicity through Oxidative Stress-Associated Pathways. *Int J Mol Sci*. 2011;12(8):5213-5237. doi:10.3390/ijms12085213.
124. Andersson MA, Nikulin M, Koljalg U, et al. Bacteria, Molds, and Toxins in Water-Damaged Building Materials. *App Environ Microbiol*. 1997 Feb;63(2):387–393.
125. Cooley JD, Wong WC, Jumper CA, Straus DC. Correlation Between the Prevalence of Certain Fungi and Sick Building Syndrome. *Occ Environ Med.* 1998 Sep;55(9):579-584.
126. Zhou X, Zhao A, Goping G, Hirszel P. Gliotoxin-Induced Cytotoxicity Proceeds via Apoptosis and is Mediated by Caspases and Reactive Oxygen Species in LLC-PK1 Cells. *Toxicol Sci*. 2000 Mar;54(1):194-202.
127. Peltola J, Andersson MA, Haahtela T, et al. Toxic-Metabolite-Producing Bacteria and Fungus in an Indoor Environment. *Appl Environ Microbiol*. 2001;67(7):3269-3274. doi:10.1128/AEM.67.7.3269-3274.2001.
128. Jussila J, Pelkonen J, Kosma VM, et al. Systemic Immunoresponses in Mice after Repeated Exposure of Lungs to Spores of *Streptomyces californicus*. *Clin Vaccine Immunol*. 2003;10(1):30-37. doi:10.1128/CDLI.10.1.30-37.2003.
129. Schwab CJ, Straus DC. Roles of *Penicillium* and *Aspergillus* in Sick Building Syndrome. *Adv Applied Microbiol*. 2004;55:215-238. doi:10.1016/S0065-2164(04)55008-6.
130. Bunger J, Westphal G, Monnich A, Hinnendahl B, Hallier E, Muller M. Cytotoxicity of Occupationally and Environmentally Relevant Mycotoxins. *Toxicology*. 2004 Oct. 1;202(3):199-211. doi:10.1016/j.tox.2004.05.007.
131. Murtoniemi T, Penttinen P, Nevalainen A, Hirvonen MR. Effects of Microbial Cocultivation on Inflammatory and Cytotoxic Production of Spores. *Inhal Toxicol*. 2005 Nov;17(12):681-693. doi:10.1080/08958370500189669.

132. Rand TG, Giles S, Flemming J, Miller JO, Puniani E. Inflammatory and Cytotoxic Responses in Mouse Lungs Exposed to Purified Toxins from Building Isolated *Penicillium brevicompactum* D*ierckx* and *P. chrysogenum* Thom. *Toxicol Sci*. 2005 Sep;87(1):213-222. doi:10.1093/toxsci/kfi223.
133. Pestka JJ, Yike I, Dearborn DG, Ward MD, Harkema JR. *Stachybotrys chartarum*, Trichothecene Mycotoxins, and Damp Building-Related Illness: New Insights into a Public Health Enigma. *Toxicol Sci*. 2008 Jul;104(1):4-26. doi:10.1093/toxsci/kfm284.
134. Corps KN, Islam Z, Pestka JJ, Harkema JR. Neurotoxic, Inflammatory, and Mucosecretory Response in the Nasal Airways of Mice Repeatedly Exposed to the Macrocyclic Trichothecene Mycotoxin Roridin A: Dose-Response and Persistence of Injury. *Toxicol Pathol.* 2010 Apr;38(3):429-451. doi:10.1177/0192623310364026.
135. Inamdar AA, Masurekar P, Hossain M, Richarson JR, Bennett JW. Signaling Pathways Involved in 1-Octet-3-ol-Mediated Neurotoxicity in *Drosophila melanogaster*: Implication in Parkinson's Disease. *Neurotox Res. 2014 Feb;25(2):183–191. doi:10.1007/s12640-013-9418-z* 1.
136. Korkalainen M, Taubel M, Naarala J, et al. Synergistic Proinflammatory Interactions of Microbial Toxins and Structural Components Characteristic to Moisture-Damaged Buildings. *Indoor Air*. 2017 Jan;27(1):13-23. doi:10.1111/ina.12282.
137. Despot DJ, Kocsube S, Bencsik O, et al. Species Diversity and Cytotoxic Potency of Airborne Sterigmatocystin-producing Aspergilli from the Section Versicolores. *Sci Total Environ*. 2016 Aug 15;562(15):296-304. doi:10.1016/j.scitotenv.2016.03.183.
138. Harding CF, Ryberg K, Biegon A. Exposure to Environmental Mold Causes Hippocampal Inflammation and Memory Loss. *Brain Behav Immun*. 2011;25(2):S186. doi:10.1016/j.bbi.2011.07.029.
139. Harding CF, Ryberg K, Pytte C. Nagai M, Ali B, Denisova K. Environmental Mold, Brain Inflammation and Memory Deficits. *Brain Behav Immun*. 2012;26(1):S47. https://doi.org/10.1016/j.bbi.2012.07.192.
140. Harding C, Liao D, Persaud R, Lin K, Page K, Pytte C. Environmental Mold Exposure, Brain Inflammation, and Spatial Memory Deficits. *Brain Behav Immun*. 2015 Oct;49(Suppl):e42. https://doi.org/10.1016/j.bbi.2015.06.160.
141. Singer R. Clinical Evaluation of Suspected Mold Neurotoxicity. Bioaerosols, Fungi, Bacteria, Mycotoxins and Human Health. Health Effects II: Toxiciology and Neurological Effects. Published by the Fungal Research Group Foundation. 2005;78-84.
142. Gordon WA, Cantor JB. The Diagnosis of Cognitive Impairment Associated with Exposure to Mold. *Adv Appl Microbiol*. 2004;55:361-374. doi:10.1016/S0065-2164(04)55014-1.
143. Gordon WA, Cantor J, Charatz H, Ashman T, Johanning E. The Chronicity of Cognitive Impairment Associated with Exposure to Toxic Mold. Bioaerosols, Fungi, Bacteria, Mycotoxins and Human Health. Health Effects II: Toxiciology and Neurological Effects. Published by the Fungal Research Group Foundation. 2005;85-91.
144. Norbäck D, Hashim JH, Cai G-H, et al. Rhinitis, Ocular, Throat and Dermal Symptoms, Headache and Tiredness among Students in Schools from Johor Bahru, Malaysia: Associations with Fungal DNA and Mycotoxins in Classroom Dust. *PLoS One*. 2016 Feb 1:1-15. doi:10.1371/journal.pone.0147996.
145. Commonwealth of Massachusetts: Special Legislative Commission on Indoor Air Pollution. Indoor Air Pollution in Massachusetts. Published April 1989.
146. U.S. Environmental Protection Agency (EPA). Report for Congress on Indoor Air Quality. Volume II: Assessment and Control of Indoor Air Pollution. August 1989.
147. Seo S, Han Y, Kim J, Choung JT, Kim BJ, Ahn K. Infrared Camera-Proven Water-Damaged Homes are Associated with the Severity of Atopic Dermatitis in Children. *Ann Allergy Asthma Immunol*. 2014 Nov;113(5):549-555. doi:10.1016/j.anai.2014.08.013.
148. Adhikari M, Negi B, Kaushik N, et al. T-2 Mycotoxin: Toxicological Effects and Decontamination Strategies. *Oncotarget*. 2017 May 16; 8(20):33933–33952.
149. Chaudhary M, Rao PV. Brain Oxidative Stress after Dermal and Subcutaneous Exposure of T-2 Toxin in Mice. *Food Chem Toxicol*. 2010;48:3436–3442. doi:10.1016/j.fct.2010.09.018.
150. Chattopadhyay P, Islam J, Goyary D, et al. Subchronic Dermal Exposure to T-2 Toxin Produces Cardiac Toxicity in Experimental Wistar Rats. *Toxicol Ind Health*. 2013;32(3):485-492. https://doi.org/10.1177/0748233713503373.
151. Muzi G, dell'Omo M, Abbritti G, Accattoli P, Fiore MC, Gabrielli AR. Objective Assessment of Ocular and Respiratory Alterations in Employees in a Sick Building. *Amer J Indust Med*. July 1998 Jul; 34(1):79-88. https://doi.org/10.1002/(SICI)1097-0274(199807)34:1<79::AID-AJIM11>3.0.CO;2-1.

152. Saeki Y, Kadonosono K, Uchio E. Clinical and Allergological Analysis of Ocular Manifestations of Sick Buildings Syndrome. *Clin Ophthalmol*. 2017;11:517-522. doi:10.2147/OPTH.S124500.
153. David J. Educate Patients about Tear-Film Stability and Quality Daily-Replacement Contact Lenses. n.d. http://reviewob.com/educate-patients-about-tear-film-stability-and-quality-daily-replacement-contact-lenses/.
154. Omar RF, Gelboin HV, Rahimtula AD. Effect of Cytochrome P450 Induction on the Metabolism and Toxicity of Ochratoxin A. *Biochem Pharmacol*. 1996 Feb 9;51(3):207-216.
155. Chen SY, Chen CJ, Tsi WY, et al. Associations of Plasma Aflatoxin B1-albumin Adduct Level with Plasma Selenium Level and Genetic Polymorphism of Glutathione S-transferases M1andT1. *Nutr Cancer*. 2000:38(2):79-85. doi:10.1207/S15327914NC382_6.
156. Sun CA, Wang LY, Chen CJ, et al. Genetic Polymorphisms of Glutathione S-transferases M1 and T1 Associated with Susceptibility to Aflatoxin-Related Heptocarcinogenesis among Chronic Hepatitis B Carriers: A Nested Case-Control Study in Taiwan. *Carcinogenesis*. 2001 Aug;22(8):1289-1294.
157. Nebert DW, Russell DW. Clinical Importance of the Cytochromes P450. *The Lancet*. 2002 October 12; 360(9340):1155-1162. doi:10.1016/S0140-6736(02)11203-7.
158. Shoemaker RC, Maizel MS. Innate Immunity, MR Spectroscopy, HLA DR, TGF beta-1, VIP and Capillary Hypoperfusion Define Acute and Chronic Human Illness Acquired Following Exposure to Water-Damaged Buildings. n.d. https://www.survivingmold.com/docs/Resources/Shoemaker%20Papers/213v1.pdf.
159. Kelada SN, Eaton DL, Wang SS, Rothman NR, Khoury MJ. The Role of Genetic Polymorphisms in Environmental Health. *Environ Health Perspect*. 2003 Jun;111(8):1055-1064.
160. Gunn, SR, Gunn GG, Mueller FW. Reversal of Refractory Ulcerative Colitis and Chronic Fatigue Syndrome Symptoms Arising from Immune Disturbance in an HLADR/DQ Genetically Susceptible Individual with Multiple Biotoxin Exposures. *Am J Case Rep*. 2016 May 11;17:320-325.
161. Guilford FT, Hope J. Deficient Glutathione in the Pathophysiology of Mycotoxin-Related Illness. *Toxins*. 2014;6:608-623. doi:10.3390/toxins6020608.
162. Eiser AR. Why does Finland have the highest dementia mortality rate? Environmental factors may be generalizable. *Brain Res*. 2017 Sep 15;1671:14-17. https://doi.org/10.1016/j.brainres.2017.06.032.
163. Hassan AA, Howayda ME, Mansour MK, et al. Prevalence of Ochratoxigenic Fungi and Ochratoxin A Residues in Animal Feeds and Modulation of their Toxic Effects by Glutathione. *Int J Curr Microbiol App Sci*. 2018;7(4):2559-2582. https://doi.org/10.20546/ijcmas.2018.704.293.
164. Al-Ansari NAA, Majeed EMA, Al-Maliky HA. Analysis of Glutathione S-Transferase M1 and T1 Polymorphism in Samples of Iraqi Children with Autism. *Mustansiriya Medical Journal*. 2017;16(3):28-38. https://doi.org/10.26903/mmj.v16i3.107.
165. Genuis SJ, Kyrillos E. The Chemical Disruption of Human Metabolism. *Toxicol Mech Methods*. 2017;27(7)1-24. https://doi.org/10.1080/15376516.2017.1323986.
166. Sears ME, Genuis SJ. Environmental Determinants of Chronic Disease and Medical Approaches: Recognition, Avoidance, Supportive Therapy, and Detoxification. *J Environ Public Health*. 2012;Article 356798:15 pages. doi:10.1155/2012/356798.
167. Bachert B, Bourdin A, Chanez P. The Nose and Sinuses in Respiratory Disorders: ERS Monograph. *Europ Respir Soc*. 2017 June 1:224 pages.
168. Wang X, Jin C, Zhong Y, et al. Glutathione (GSH) Reduces Cytotoxicity Evoked by Patulin (PAT) in HEK293 Cells by Preventing Oxidative Damage and Mitochondrial Apoptotic Pathway. *J Agric Food Chem*. 2018 Apr 20. doi:10.1021/acs.jafc.8b01212.
169. Sadiq IZ, Pankaj T, Khan AR, Naziru D, Safiyanu I, Salisu AR. Cytoprotective, Conjugative and Antioxidant Activities of Glutathione; and Its Role in Removal of Toxic Metabolites and Protein Protection: A Review. *Chem Res J*. 2016;1(4):147-153.
170. Townsend DM, Tew KD, Tapiero H. The Importance of Glutathione in Human Disease. *Biomedicine & Pharmacotherapy*. 2003 May;57(3–4):145-155. https://doi.org/10.1016/S0753-3322(03)00043-X
171. Wang JE, Warris A, Ellingsen EA, et al. Involvement of CD14 and Toll-Like Receptors in Activation of Human Monocytes by *Aspergillus fumigatus* Hyphae. *Infect Immun*. 2001 Apr;69(4):2402-2406. doi:10.1128/IAI.69.4.2402-2406.2001.
172. Lehnardt S, Lachance C, Patrizi S, et al. Toll-Like Receptor TLR4 is Necessary for Lipopolysaccharide-Induced Oligodendrocyte Injury in the CNS. *J Neurosci*. 2002 Apr 1;22:2478-2486. doi:20026268.

173. Meier A, Kirschning CJ, Nikolaus T, Wagner H, Heesemann J, Ebel F. Toll-like receptor (TLR2) and TLR4 are Essential for *Aspergillus*-induced Activation of Murine Macrophages. *Cell Microbiol*. 2003 Aug;5:561-570.
174. Moretti S, Bellocchio S, Bonifazi P, Bozza S, Zelante T, Bistoni F, Romani L. The Contribution of PARs to Inflammation and Immunity to Fungi. *Mucosal Immunol*. 2008 Mar;1(2):156-168. doi:10.1038/mi.2007.13.
175. Roeder A, Kirschning C, Rupec R, Schaller M, Weindl G, Korting H. Toll-like Receptors as Key Mediators in Innate Antifungal Immunity. *Med Mycol*. 2004 Dec;42(6):485-498.
176. Netea MG, Van der Graaf C, Van der Meer JW, Kullberg BJ. Recognition of Fungal Pathogens by Toll-Like Receptors. *Eur J Clin Microbiol Infect Dis*. 2004 Sep;23(9):672-6. doi:10.1007/s10096-004-1192-7.
177. Beurel E. A Primer on Inflammation for Psychiatrists. *Psychiatr Ann*. 2015 May;45(5):226-231. doi:10.3928/00485713-20150501-04.
178. Kankkunen P, Rintahaka J, Aalto A, et al. Trichothecene Mycotoxins Activate Inflammatory Response in Human Macrophages. *J Immunol*. 2009 May 15;182(10):6418-6425. doi:10.4049/jimmunol.0803309.
179. Murtoniemi T, Nevalainen A, Suutari M, Toivola M, Komulainen H, Hirvonen MR. Induction of Cytotoxicity and Production of Inflammatory Mediators in RAW264.7 Macrophages by Spores Grown on Six Different Plasterboards. *Inhal Toxicol*. 2001;13(3):233-247. https://doi.org/10.1080/08958370150502467.
180. Cunningham C, Wilcockson DC, Campion S, Lunnon K, Perry VH. Central and Systemic Endotoxin Challenges Exacerbate the Local Inflammatory Response and Increase Neuronal Death During Chronic Neurodegeneration. *J Neurosci*. 2005 Oct 5;25(40):9275-9284. doi:10.1523/JNEUROSCI.2614-05.2005.
181. Hirotsu M, Shiozawa A, Ono N, et al. Fungal Extracts Detected in Eosinophilic Chronic Rhinosinusitis Induced Cytokines from the Nasal Polyp Cells. *The Laryngoscope*. 2014.124:E347-E353. doi:10.1002/lary.24655.
182. Wolf CHJ. Innate Immunity and the Pathogenicity of Inhaled Microbial Particles. *Int J Biol Sci*. 2011;7(3):261-268.
183. Gorny RL. Filamentous Microorganisms and Their Fragments in Indoor Air - A Review. *Ann Agric Environ Med*. 2004;11(2):185-197.
184. Gorny RL, Mainelis G, Grinshpun SA, Willeke K, Dutkiewicz J, Reponen T. Release of *Streptomyces albus P*ropagules from Contaminated Surfaces. *Environ Res*. 2003 Jan;91(1):45-53.
185. Gorny RL, Reponen T, Willeke K, et al. Fungal Fragments as Indoor Air Biocontaminants. *Appl Environ Microbol*. 2002;68(7)3552-3531. doi:10.1128/AEM.68.7.3522-3531.2002.
186. Reponen T, Sung-Chul S, Grimsley F, Lee T, Crawford C, Grinshpun SA. Fungal Fragments in Moldy Houses: A Field Study in Homes in New Orleans and Southern Ohio. *Atmos Environ*. 2007 Dec;41(37):8140-8149.
187. Straus DC. Molds, Mycotoxins and Sick Building Syndrome. *Toxicol Ind Health*. 2009;25(9-10):617-635. doi:10.1177/0748233709348287.
188. Gao P, Martin J. Volatile Metabolites by Three Strains of *Stachybotrys chartarum* Cultivated on Rice and Gypsum Board. *Appl Occup Environ Hyg*. 2002 Jun;17(6):430-6. doi:10.1080/10473220290035462.
189. Torvinen E, Meklin T, Torkko P, et al. 2006. *Mycobacteria* in Moisture-Damaged Building Materials. *Appl Environ Microbiol*. 2006 Oct;72(10):6822-6824. doi:10.1128/AEM.00588-06.
190. Taubel M, Sulyok M, Vishwanath V, et al. Co-Occurrence of Toxic Bacterial and Fungal Secondary Metabolites in Moisture-Damaged Indoor Environments. *Indoor Air*. 2011 Oct;21(5):368-375. doi:10.1111/j.1600-0668.2011.00721.x.
191. Peitzsch M, Sulyok M, Taubel M, et al. Microbial Secondary Metabolites in School Buildings Inspected for Moisture Damage in Finland, The Netherlands and Spain. *J Environ Monit*. 2012 Aug;14(8):2044-2053. doi:10.1039/c2em30195d.
192. Shoemaker RC, Mark L, McMahon SW, Thrasher JD, Grimes C. Research Committee Report on Diagnosis and Treatment of Chronic Inflammatory Response Syndrome Caused by Exposure to the Interior Environment of Water-Damaged Buildings. Published by the Policyholders of America (POA). July 27, 2010.
193. Berndtson K, McMahon S, Ackerley M, Rapaport S, Gupta S, Shoemaker RC. Consensus Statement, Part 1: Medically Sound Investigation and Remediation of Water-Damaged Buildings in Cases of CIRS-WDB. October 30, 2015. https://www.survivingmold.com/MEDICAL_CONSENSUS_STATEMENT_10_30_15.PDF.
194. Vetter P, Rossi L, Edwards C. A Recovery Manual for Patients and Families Impacted by CIRS. Mold Illness: Surviving and Thriving. Published by BookBaby. January 30, 2018.
195. Shoemaker RC, House D, Ryan JC. Vasoactive Intestinal Polypeptide (VIP) Corrects Chronic Inflammatory Response Syndrome (CIRS) Acquired Following Exposure to Water-Damaged Buildings. *Health*. 2013;5(3):396-401. Article ID:28586,6 pages. doi:10.4236/health.2013.53053.

196. Eskandari F, Webster JI, Sternberg EM. Neural Immune Pathways and Their Connection to Inflammatory Diseases. *Arthritis Res Ther*. 2003;5(6):251-265. doi:10.1186/ar1002.
197. Watkins LR, Maier SF. Immune Regulation of Central Nervous System Functions: From Sickness Responses to Pathological Pain. *J Intern Med*. 2005;257(2):139-155. doi:10.1111/j.1365-2796.2004.01443.x.
198. Hopkins SJ. Central Nervous System Recognition of Peripheral Inflammation: A Neural, Hormonal Collaboration. *Acta Biomed*. 2007;78(Suppl 1):231-247.
199. Wilson CJ, Finch CE, Cohen HJ. Cytokines and Cognition: The Case for a Head-to-Toe Inflammatory Paradigm. *J Am Geriatr Soc*. 2002 Dec;50(12):2041-2056.
200. Iwamoto S, Iwai S-i, Tsujiyama K, et al. TNF-alpha Drives C14+ Monocytes to Differentiate into CD70+ Dendritic Cells Evoking Th1 and Th17 Responses. *J Immunol*. 2007;179:1449-1457.
201. Zelante T, De Luca A, Bonifazi P, et al. IL-23 and the Th17 Pathway Promote Inflammation and Impair Antifungal Immune Resistance. *Europ J Immunol*. 2007 Oct;37(10):2695–2706. doi:10.1002/eji.200737409.
202. Vojdani A, Lambert J. The Role of Th17 in Neuroimmune Disorders: Target for CAM Therapy. Part I. *Evid Based Complementary Alternat Med*. 2011;Article ID 927294:8pages. doi:10.1093/ecam/nep062.
203. Vojdani A, Lambert J The Role of Th17 in Neuroimmune Disorders: Target for CAM Therapy. Part II. *Evid Based Complement Alternat Med*. 2011;Article ID 984965:7 pages. https://doi.org/10.1093/ecam/nep063.
204. Vojdani A, Lambert J, Kellermann G. The Role of Th17 in Neuroimmune Disorders: A Target for CAM Therapy. Part III. *Evid Based Complement Alternat Med*. 2011;Article ID 548086. doi:10.1093/ecam/nep064.
205. Valdes JJ, Cameron JE, Cole RJ. Aflatrem: A Tremorgenic Mycotoxin with Acute Neurotoxic Effects. *Environ Health Perspect*. 1985 Oct;62:459–463.
206. Islam Z, Amuzie CJ, Harkema JR, Pestka JJ. Neurotoxicity and Inflammation in the Nasal Airways of Mice Exposed to Macrocyclic Trichothecene Mycotoxin Roridin: A Kinetics and Potentiation by Bacterial Polysaccharide Coexposure. *Toxicol Sci*. 2007 Aug 1;98(2):526–541. https://doi.org/10.1093/toxsci/kfm102.
207. Doi K, Uetsuka K. Mechanisms of Mycotoxin-Induced Neurotoxicity and Tumorigenesis through Oxidative Stress-Associated Pathways. *Int J Mol Sci*. 2014;27(1):1-10. https://doi.org/10.1293/tox.2013-0062.
208. Rafnsson SB, Deary IJ, Smith FB, et al. Cognitive Decline and Markers of Inflammation and Hemostasis: The Edinburgh Artery Study. *J Am Geriatr Soc*. 2007;55(5):700-707. https://doi.org/10.1111/j.1532-5415.2007.01158.x.
209. Jedrychowski W, Maugeri U, Perera F, et al. Cognitive Function of 6-year-old Children Exposed to Mold-Contaminated Homes in Early Postnatal Period. Prospective Birth Cohort Study in Poland. *Physiol Behav*. 2011 Oct 24;104(5):989-995. https://doi.org/10.1016/j.physbeh.2011.06.019.
210. Perry VH. The Influence of Systemic Inflammation on Inflammation in the Brain: Implications for Chronic Neurodegenerative Disease. *Brain Behav Immun*. 2004 Sep;18(5):407-413. https://doi.org/10.1016/j.bbi.2004.01.004.
211. Vojdani A, Campbell AW, Kashanian A, Vojdani E. Antibodies Against Molds and Mycotoxins Following Exposure to Toxigenic Fungi in a Water-Damaged Building. *Arch Environ Health*. 2003 Jun;58(6):324-36.
212. Shoemaker RC, House D, Ryan JC. Structural Brain Abnormalities in Patients with Inflammatory Illness Acquired Following Exposure to Water-Damaged Buildings: A Volumetric MRI Study Using NeuroQuant, *Neurotoxicol Teratol*. 2014 Sep-Oct; 45: 18-26.
213. Calderon-Garciduenas L, Mora-Tiscareno A, Ontiveros E, et al. Air Pollution, Cognitive Deficits and Brain Abnormalities: A Pilot Study with Children and Dogs. *Brain Cogn*. 2008 Nov;68(2):117-127. https://doi.org/10.1016/j.bandc.2008.04.008.
214. Ackerley M. Brain on Fire: The Role of Toxic Mold Triggering Psychiatric Symptoms. March 24, 2014.
215. Trout D, Bernstein J, Martinez K, Biagini R, Wallingford K. Bioaerosol Lung Damage in a Worker with Repeated Exposure to Fungi in a Water-Damaged Building. *Environ Health Perspect*. 2001 Jun;109(6):641-644.
216. McMahon SW, Shoemaker RC, Ryan JC. Reduction in Forebrain Parenchymal and Cortical Grey Matter Swelling Across Treatment Groups in Patients with Inflammatory Illness Acquired Following Exposure to Water-Damaged Buildings. *J Neurosci Clin Res*. 2016 Mar 21;1(1). doi:10.4172/jnscr.1000102.
217. Pisa D, Alonso R, Jimenez-Jimenez FJ, Carrasco L. Fungal Infection in Cerebrospinal Fluid from Some Patients with Multiple Sclerosis. *Eur J Clin Microbiol Infect Dis*. 2013;32:795–801. doi:10.1007/s10096-012-1810-8.
218. Qin L, Liu Y, Wang T, et al. NADPH Oxidase Mediates Lipopolysaccharide-Induced Neurotoxicity and Proinflammatory Gene Expression in Activated Microglia. *J Biol Chem*. 2004;279(2):1415-1421. doi:10.1074/jbc.M307657200.

219. Browne SE, Lin L, Mattsson A, Georgievska B, Isacson O. Selective Antibody-Induced Cholinergic Cell and Synapse Loss Produce Sustained Hippocampal and Cortical Hypometabolism with Correlated Cognitive Deficits. *Exp Neurol*. 2001 Jul;170(1):36-47. https://doi.org/10.1006/exnr.2001.7700.
220. Kidd PM. Attention Deficit/Hyperactivity Disorder (ADHD) in Children: Rationale for its Integrative Management. *Alt Med Rev*. 2000;5(5):402-428.
221. Crago BR, Nelson LA. QEEG and Neuropsychological Consequences of Exposure to Toxic Molds. *J Neurotherapy*. 2004;8:142.
222. Costa LG, Manzo L. Occupational Neurotoxicology. Published by CRC Press. May 18, 1998.
223. Saari A, Tolonen U, Paakko E, et al. Cardiovascular Autonomic Dysfunction Correlates with Brain MRI Lesion Load in MS. *Clin Neurophysiol*. 2004 Jun;115(6):1473-1478. https://doi.org/10.1016/j.clinph.2004.01.012.
224. Neuro-Optometric Rehabilitation: A Specialty Worth Examining. Interview with Dr. William V. Padula. *Optometric Management*. November 1, 2009.
225. Padula, WV. Mild Traumatic Brain Injury (mTBI): Symptoms without Evidence. *J Neurol Sci*. 2016 Nov 15;370:303-304. https://doi.org/10.1016/j.jns.2016.09.015.
226. Giuseppe V, Fazio C, Milone S, Blandino A, Salvi L, Messina C. Cardiovascular Autonomic Dysfunction in Multiple Sclerosis is Likely Related to Brainstem Lesions. *J Neurol Sci*. 1993 Dec;120(1):82-86. https://doi.org/10.1016/0022-510X(93)90029-X.
227. Pazo JH, Belforte JE. Basal Ganglia and Functions of the Autonomic Nervous System. *Cell Mol Neurol*. 2002 Dec;22(5-6):645-654. https://doi.org/10.1023/A:1021844605250.
228. Peraica M, Radic B, Lucic A, Pavlovic M. Toxic Effects of Mycotoxins in Humans. *Bulletin of the World Health Organization*. 1999;77(9):754-766.
229. Breuel KF, Kougias P, Rice PJ, et al. Anterior Pituitary Cells Express Pattern Recognition Receptors for Fungal Glucans: Implications for Neuroendocrine Immune Involvement in Response to Fungal Infections. *Neuroimmunomodulation*. 2004;11(1):1-9. https://doi.org/10.1159/000072963.
230. Shoemaker RC, Rash JM, Simon EW. Sick Building Syndrome in Water-Damaged Buildings: Generalization of the Chronic Biotoxin-Associated Illness Paradigm to Indoor Toxigenic Fungi. Published in E. Johanning (Ed.), Bioaerosols, Fungi, Bacteria, Mycotoxins and Human Health: Pathophysiology, Clinical Effects, Exposure Assessment, Prevention and Control in Indoor Environments and Work, Fungal Research Group Foundation Inc., Albany, NY, 2005, pp. 66–77.
231. Landman, RE, Puder, JJ, Xiao E, Freda PU, Ferin M, Wardlaw SL. Endotoxin Stimulates Leptin in the Human and Nonhuman Primate. *J Clin Endocrinol Metab*. 2003;88(3):1285-1291. https://doi.org/10.1210/jc.2002-021393.
232. Considine RV, Sinha MK, Heiman ML, et al. Serum Immunoreactive-Leptin Concentrations in Normal-Weight and Obese Humans. *N Engl J Med*. 1996 Feb;334(5):292-295. doi:10.1056/NEJM199602013340503.
233. Williams KW, Scott MM, Elmquist JK. From Observation to Experimentation: Leptin Action in the Mediobasal Hypothalamus. *Am J Clin Nutr.* 2009 Mar 1;89(3):985S-990S. https://doi.org/10.3945/ajcn.2008.26788D.
234. Heiman ML, Ahima RS, Craft LS, Schoner B, Stephens TW, Flier JS. Leptin Inhibition of the Hypothalamic-Pituitary-Adrenal Axis in Response to Stress. *Endocrinology*. 1997 Sep 1;138(9):3859-3863. https://doi.org/10.1210/endo.138.9.5366.
235. Hamilton BS, Paglia D, Kwan AYM, Deitel M. Increased Obese mRNA Expression in Omental Fat Cells from Massively Obese Humans. *Nat Med*. 1995 Sep;1(9):953-956. doi:10.1038/nm0995-953.
236. Shoemaker RC, Johnson K, Lysander J, et al. Diagnostic Process for Chronic Inflammatory Response Syndrome (CIRS): A Consensus Statement Report of the Consensus Committee of Surviving Mold. *Int Med Rev*. 2018 May;4(5).
237. Somppi TL. Non-Thyroidal Illness Syndrome in Patients Exposed to Indoor Air Dampness Microbiota Treated Successfully with Triiodothyronine. *Front. Immunol*. 2017 Aug 07;8(Article 919):1-8. https://doi.org/10.3389/fimmu.2017.00919.
238. National Academy of Hypothyroidism. Understanding Local Control of Thyroid Hormones:(Deiodinases Function and Activity). n.d. https://www.nahypothyroidism.org/deiodinases/.
239. Holck P, Sletmoen M, et al. Potentiation of Histamine Release by Microfungal (1→3)- and (1→6)-β-D-Glucans. *Basic Clin Pharmacol Toxicol*. 2007 Dec;101(6):455-458. https://doi.org/10.1111/j.1742-7843.2007.00140.x.
240. Anyanwu EC, Morad M, Campbell AW. Metabolism of Mycotoxins, Intracellular Functions of Vitamin B12, and Neurological Manifestations in Patients with Chronic Toxigenic Mold Exposures. A Review. *Scientific World Journal*. 2004 Aug 26;4:736-745. http://dx.doi.org/10.1100/tsw.2004.133.

241. Haas H, Sergeva OA, Selbach O. Histamine in the Nervous System. *Physiol Rev*. 2008;88(3):1183-1241. https://doi.org/10.1152/physrev.00043.2007.
242. Lander F, Meyer HW, Norm S. Serum IgE Specific to Moulds, Measured by Basophil Histamine Release, is Associated with Building-Related Symptoms in Damp Buildings. *Inflamm. Res*. 2001;50:227–231.
243. Tan B, Yin Y, Kong X, et al. L-Arginine Stimulates Proliferation and Prevents Endotoxin-Induced Death of Intestinal Cells. *Amino Acids*. 2010 Apr;38(4)1227-1235.
244. Zhu HL, Liu YL, Xie XL, et al. Effect of L-Arginine on Intestinal Mucosal Immune Barrier Function in Weaned Pigs after *Escherichia coli* LPS Challenge. *Innate Immun*. 2013;19(3):242-252. https://doi.org/10.1177/1753425912456223.
245. Vinitsky AR, Parks RR. Bipolar Illness: An Environmental and Nutritional Approach to Therapy. Chapter 32 in Advancing Medicine with Food and Nutrition, 2nd Edition. 2012 Dec 10;595-614.
246. Liu Y, Han J, Huang J, Wang X, Wang F, Wang J. Dietary L-Arginine Supplementation Improves Intestinal Function in Weaned Pigs after an *Escherichia coli* Lipopolysaccharide Challenge. *Asian-Australian Journal of Animal Sciences*. 2009;22(12):1667-1675. doi:https://doi.org/10.5713/ajas.2009.90100.
247. Theoharides TC, Donelan JM, Papadopoulou N, Cao J, Kempuraj D, Conti P. Mast Cells as Targets of Corticotropin-Releasing Factor and Related Peptides. *Trends in Pharmacol Sci*. 2004 Nov;25(11):563-568. https://doi.org/10.1016/j.tips.2004.09.007.
248. Theoharides TC, Kalogeromitros D. The Critical Role of Mast Cells in Allergy and Inflammation. *Annals New York Acad Sci*. 2006 Nov;1088(1):78-99. https://doi.org/10.1196/annals.1366.025.
249. Carnahan J. Mold is a Major Trigger of Mast Activation Cell Syndrome. 2018. https://www.jillcarnahan.com/2018/03/12/mold-is-a-major-trigger-of-mast-activation-cell-syndrome/.
250. Carnahan J. Five Essential Tips for Living with Mold Toxicity and CIRS. 2018. https://www.jillcarnahan.com/2018/03/26/five-essential-tips-for-living-with-mold-toxicity-and-cirs/.
251. Rea WJ. What Primary Physicians Should Know About Environmental Causes of Illness. *AMA J Ethics*. 2009 Jun;11(6):473-476.
252. Small BM, Eng P. Indoor Air Pollutants in Residential Settings: Respiratory Health Effects and Remedial Measures to Minimize Exposure. *A Review for the Ontario Lung Association*. April 2002.
253. Brunekreef B, Dockery D, Speizer FE. Home Dampness and Respiratory Morbidity in Children. *Am Rev Resp Dis*. 1989;140(5):1363-1367. https://doi.org/10.1164/ajrccm/140.5.1363.
254. Dales R, Zwanenburg H, Burnett R, Franklin C. Respiratory Health Effects of Home Dampness and Molds Among Canadian Children. *Am J Epidemiol*. 1991;134(2):196-203. https://doi.org/10.1093/oxfordjournals.aje.a116072.
255. Zock J, Jarvis D, Luczynska C, Sunyer J, Burney P. Housing Characteristics, Reported Mold Exposure, and Asthma in the European Community Respiratory Health Survey. *J Allergy Clin Immunol*. 2002;110(2):285-292. https://doi.org/10.1067/mai.2002.126383.
256. Strachan DP, Flannigan B, McCabe E, McGarry F. Quantification of Airborne Moulds in the Homes of Children with and without Wheeze. *Thorax*. 1990;45:382-387. http://dx.doi.org/10.1136/thx.45.5.382.
257. Brunekreef B. Damp Housing and Adult Respiratory Symptoms. *Allergy*. 1992;47(5):498-502. https://doi.org/10.1111/j.1398-9995.1992.tb00672.x.
258. Waegemaekers M, van Wageningen N, Brunekreef B, Boleij JSM. Respiratory Symptoms in Damp Homes. A Pilot Study. *Allergy*. 1989;44(3):192-198. https://doi.org/10.1111/j.1398-9995.1989.tb02261.x.
259. Jaakkola J, Jaakola N, Ruotsalainen R. Home Dampness and Molds as Determinants of Respiratory Symptoms and Asthma in Pre-School Children. *J Exposure Anal Environ Epidemiol*, 1993 Jan 1;3(Suppl 1):126-142.
260. Walinder R, Ernstgård L, Johanson G, Norbäck D, Venge P, Wieslander G. Acute Effects of a Fungal Volatile Compound. *Environ Health Perspect*. 2005 Dec;113(12):1775-1778.
261. Hernberg S, Sripaiboonkij P, Quansah R, Jaakkola JJK, Jaakkola MS. Indoor Molds and Lung Function in Healthy Adults. *Respir Med*. 2014 May;108(5):677-684. https://doi.org/10.1016/j.rmed.2014.03.004.
262. Falkinham JO III. *Mycobacterial* Aerosols and Respiratory Disease. *Emerg Infect Dis*. 2003 Jul;9(7):763-7. doi:10.3201/eid0907.02-0415.
263. Iossifova YY, Cox-Ganser JM, Park JH, White SK, Kreiss K. Lack of Respiratory Improvement Following Remediation of a Water-Damaged Office Building. *Am J Ind Med*. 2011;54(4):269-277. https://doi.org/10.1002/ajim.20910.

264. Oluwole O, Rennie DC, Senthilselvan A, et al. The Association Between Endotoxin and beta-(1->3)-D Glucan in House Dust with Asthma Severity Among Schoolchildren. *Resp Med*. 2018 May;138:38-46. https://doi.org/10.1016/j.rmed.2018.03.015.
265. Byeon JH, Ri S, Amarsaikhan O, et al. Association between Sensitization to Mold and Impaired Pulmonary Function in Children with Asthma. *Allergy Asthma Immunol Res*. 2017 Nov;9(6):509-516. https://doi.org/10.4168/aair.2017.9.6.509
266. Gray MR, Thrasher JD, Hooper D, Crago R. A Case of Reyes-Like Syndrome in a 68-Day-Old Infant: Water Damaged Home, Mold, Bacteria and Aflatoxins. *Int J Toxicol*. 2015;2(2):42-54.
267. Park JH, Cox-Ganser JM, White SK, et al. Bacteria in a Water-Damaged Building: Associations of Actinomycetes and Non-Tuberculous Mycobacteria with Respiratory Health in Occupants. *Indoor Air*. 2017 Jan;27(1):24-33. https://doi.org/10.1111/ina.12278.
268. Rautiala S, Torvinen E, Torkko P, et al. Potentially Pathogenic, Slow-Growing Mycobacteria Released into Workplace Air During the Remediation of Buildings. *J Occ Environ Hygiene*. 2004;1:1-6.
269. Falkinham JO. Mycobacterial Aerosols and Respiratory Disease. *Emerg Infect Dis*. 2003;9(7):763-767.
270. Huttunen K, Ruotsalainen M, Iivanainen T, Torkko P, Katila ML, Hirvonen MR. Inflammatory Reponses in RAW264.7 Macrophages Caused by Mycobacteria Isolated from Moldy Houses. *Environ Toxicol Pharmacol*. 2000;8:237-244.
271. Apostolakos MJ, Rossmoore H, Beckett WS. Hypersensitivity Pneumonitis from Ordinary Residential Exposures. *Environ Health Perspect*. 2001 Sep;109(9). http://dx.doi.org/10.1289/ehp.01109979.
272. Tibuhwa DD. Moulds Menaces in Flood-Ravaged Homes: A Case Study of Dar Es Salaam City Tanzania. *J Biol Life Sci.* 2016;7(1):110-121.
273. Ando M, Arima K, Yoneda R, Tamura M. Japanese Summer-Type Hypersensitivity Pneumonitis: Geographic Distribution, Home Environment, and Clinical Characteristics of 621 Cases. *Amer Rev Resp Dis*. 1991;144(4). https://doi.org/10.1164/ajrccm/144.4.765.
274. Knutsen AP, Bush RK, Demain JG, et al. Fungi and Allergic Lower Respiratory Tract Diseases. *J Allergy Clin Immunol*. 2012 Feb;129(2):280-191. doi:https://doi.org/10.1016/j.jaci.2011.12.970.
275. Fairs A, Agbetile J, Hargadon B, et al. IgE Sensitization to *Aspergillus fumigatus* is Associated with Reduced Lung Function in Asthma. *Amer J Resp Crit Care Med*. 2010 Dec 1;182(11). https://doi.org/10.1164/rccm.201001-0087OC.
276. Johanning E, Auger P, Morey PR, Yang CS, Olmsted E. Review of Health Hazards and Prevention Measures for Response and Recovery Workers and Volunteers After Natural Disasters, Flooding And Water Damage: Mold and Dampness. *Environ Health Prev Med*. 2013;19:368.
277. Huttunen K. Indoor Air Pollution. *Clinical Handbook of Air Pollution-Related Diseases*. 2018:107-114.
278. Hope J, Hope BE. A Review of the Diagnosis and Treatment of Ochratoxin A Inhalational Exposure Associated with Human Illness and Kidney Disease including Focal Segmental Glomerulosclerosis. *J Environ Public Health*. 2012;Article ID 835059:10 pages. http://dx.doi.org/10.1155/2012/835059.
279. Pfohl-Leszkowicz A, Manderville RA. Ochratoxin A: An Overview on Toxicity and Carcinogenicity in Animals and Humans. *Molecular Nutr Food Res*. 2007;51(1):61-99. https://doi.org/10.1002/mnfr.200600137.
280. Anyanwu E, Campbell AW, Vojdani A, Ehiri JE, Akpan IA. Biochemical Changes in the Serum of Patients with Chronic Toxigenic Mold Exposures: A Risk Factor for Multiple Renal Dysfunctions. *Sci World J*. 2003 Nov 3;3(11);1058-1064. http://dx.doi.org/10.1100/tsw.2003.92.
281. Dipaolo N, Guarnieri A, Garosi G, Sacchi G, Mangiarotti AM. Inhaled Mycotoxins Lead to Acute Renal Failure. *Nephrology Dialysis Transplantation*. 1994;9(4):116-120.
282. Dai Y, She W, Zhu W, et al. Diagnosis and Treatment of Mycotic Otitis Media. *Lin Chung Er Bi Yan Hou Tou Jing Wai Ke Za Zhi*. 2009 Jan;23(1):11-13.
283. Hamzany Y, Soudry E, Preis M, et al. Fungal Malignant External Otitis. *J Infect*. 2011 Mar;62(3):226-231. doi:10.1016/j.jinf.2011.01.001.
284. Martin TJ, Kerschner JE, Flanary VA. Fungal Causes of Otitis Externa and Tympanostomy Tube Otorrhea. *Int J Pediatr Otorhinolaryngol*. 2005 Nov;69(11):1503-1508. doi:10.1016/j.ijporl.2005.04.012.
285. Morris MC, Almudevar AL, Casey JR, Pichichero ME. Familial and Microbiological Contribution to the Otitis-Prone Condition. *Int J Pediatr Otorhinolaryngol*. 2015 Dec;79(12):2174-2177. doi:10.1016/j.ijporl.2015.09.043.
286. Shoemaker RC, Maizel MS. Exposure to Interior Environments of Water-Damaged Buildings Causes a CFS-like Illness in Pediatric Patients: A Case/Control Study. *Bulletin of the IACFS/ME*. January 2009;17(2):69-81.

287. Dennis D, Thrasher JD. Surgical and Medical Management of Sinus Mucosal and Systemic Mycotoxicosis. *Journal of Otolaryngology & Reconstructive Surgery*. 2017 Apr 24;3(1):1-7.
288. Tibuhwa DD. Moulds Menaces in Flood-Ravaged Homes: A Case Study of Dar Es Salaam City Tanzania. *J Biol Life Sci*. 2016;7(1). doi:https://doi.org/10.5296/jbls.v7i1.8681.
289. Brewer JH, Thrasher JD, Straus DC, Madison RA, Hooper D. Detection of Mycotoxins in Patients with Chronic Fatigue Syndrome. Toxins. 2013;5(4):605-17. doi:10.3390/toxins5040605.
290. Johnson E. Mold at Ground Zero for CFS. Chapter 23 in Mold Warriors: Fighting America's Hidden Health Threat, by Shoemaker RC. Published by Gateway Press. April 2005.
291. Petrison L. Back from the Edge: How One Man's Discovery Brought Him from Desperately Sick with Chronic Fatigue Syndrome to the Top of Mt. Whitney in Six Months. Published by Paradigm Change. August 6, 2013.
292. Rehmeyer J. Through the Shadowlands: A Science Writer's Odyssey into an Illness Science Doesn't Understand. Published by Rodale Books. May 23, 2017.
293. Katole SB, Pawar MM, Rahman S, Kulkarni R. Mycotoxins: How to Deal with this Problem? *Livestock Line*. 2011 Sep;5(5):21-23.
294. Hudnell HK. Chronic Biotoxin-Associated Illness: Multiple-System Symptoms, a Vision Deficit, and Effective Treatment. *Neurotoxicol Teratol*. 2005 Sep-Oct;27(5):733-743.
295. Naviaux R, Naviaux JC, Li K,et al. Metabolic Features of Chronic Fatigue Syndrome. PNAS, Proceedings of the National Academy of Sciences of the United States of America. 2016;113(37):E5472-E5480. https://doi.org/10.1073/pnas.1607571113.
296. Chronic Biotoxin-Associated Illness Paradigm to Indoor Toxigenic Fungi. E. Johanning (Ed.), Bioaerosols, fungi, bacteria, mycotoxins and human health: patho-physiology, clinical effects, exposure assessment. Health Effects II – Toxicology and Neurological Effects. 2005:52-63.
297. Shoemaker R. Mold Illness Treatment--Step by Step. https://www.survivingmold.com/treatment/step-by-step.
298. McMahon SW. An Evaluation of Alternate Means to Diagnose Chronic Inflammatory Response Syndrome and Determine Prevalence. *Med Res Arch*. 2017;5(3)1-18.
299. Sprouse A. Integrative Sexual Health, Chapter 10, Toxins: A Pervasive 21st-Century Problem for Health and Sexuality. Oxford University Press. March 14, 2018. (Sprouse treatment: mold avoidance, infusions of intravenous nutrients, sauna detoxification, heavy metal chelation)
300. Storey E, Dangman KH, Schenck, P, et al. Guidance for Clinicians on the Recognition and Management of Health Effects Related to Mold Exposure and Moisture Indoors. University of Connecticut Health Center, Division of Occupational and Environmental Medicine, Center for Indoor Environments and Health. September 30, 2004. https://health.uconn.edu/occupational-environmental/wp-content/uploads/sites/25/2015/12/mold_guide.pdf.
301. Crinnion WJ, Pizzorno JE. Clinical Environmental Medicine. Identification and Natural Treatment of Disease Caused by Common Pollutants. *Elsevier Health Science*. April 26, 2018:1423 pages.
302. Valtonen V. Clinical Diagnosis of the Dampness and Mold Hypersensitivity Syndrome: Review of the Literature and Suggested Diagnostic Criteria. *Front. Immunol*. 2017 Aug;8(Article 95.1):1-6. https://doi.org/10.3389/fimmu.2017.00951.
303. Vinitsky AR. Reversing Autonomic Nervous System Dysfunction by Potentiating Methylation. US Patent 20120231089 A1. September 13, 2012.
304. Vinitsky AR. Enlightened Medicine. https://enlightenedmedicine.net.
305. Vinitsky AR, Golos N. Energy: The Essence of Environmental Health. Published by Author House. October 13, 2004, 384 pages.
306. Rea WJ, Pan Y, Griffiths B. The Treatment of Patients with Mycotoxin-Induced Disease. *Toxicol Ind Health.* 2009;25(9-10)711-714. https://doi.org/10.1177/0748233709348281.
307. Rea W. Environmental Health Center - Dallas. https://www.ehcd.com.
308. Rea WJ, Patel KD. Reversibility of Chronic Disease and Hypersensitivity, Volume 1: Regulating Mechanisms of Chemical Sensitivity. Published by CRC Press. June 18, 2010:594 pages.
309. Rea WJ, Patel KD. Reversibility of Chronic Disease and Hypersensitivity, Volume 2: The Effects of Environmental Pollutants on the Organ System. Published by CRC Press. August 15, 2014:723 pages.
310. Rea WJ, Patel KD. Reversibility of Chronic Disease and Hypersensitivity, Volume 3: Clinical Environmental Manifestations of the Neurocardiovascular Systems. Published by CRC Press. September 26, 2014:413 pages.

311. Rea WJ, Patel KD. Reversibility of Chronic Disease and Hypersensitivity, Volume 4: The Environmental Aspects of Chemical Sensitivity. Published by CRC Press. November 22, 2017:116 pages.
312. Rea WJ, Patel KD. Reversibility of Chronic Disease and Hypersensitivity, Volume 5: Treatment Options of Chemical Sensitivity. Published by CRC Press. December 14, 2017:902 pages.
313. Rea WJ. History of Chemical Sensitivity and Diagnosis. *Rev Environ Health*. 2016 Sep 1;31(3):353-361. https://doi.org/10.1515/reveh-2015-0021.
314. Gustafson C. William Rea MD: Investigating Environmental Sensitivities May Resolve "Uncurable" Illness. *Integr Med (Encinitas)*. 2016 Mar;15(1):24-26.
315. Lieberman A. Center for Occupational & Environmental Medicine. http://www.coem.com.
316. Collegium Ramazzini. Tribute to Dr. Kaye H. Kilburn. August 7, 2014. http://www.collegiumramazzini.org/news1.asp?id=118.
317. Collegium Ramazzini. http://www.collegiumramazzini.org/about.asp.
318. Kilburn KH. Inhalation of Molds and Mycotoxins. *Eur J Oncol*. 2002;7:197-202.
319. Kilburn KH. Indoor Mold Exposure Associated with Neurobehavioral and Pulmonary Impairment: A Preliminary Report. *Arch Environ Health*. 2003 Jul;58(7):390-398. https://doi.org/10.1080/00039896.2003.11879139.
320. Ziem G. Neural Sensitization: The Medical Key to Treatment of Chemical Injury. 2013 Dec 10;21 pages.
321. Miller SP, Marinkovich, VA, Riege DH, et al. Application of the MAST Immunodiagnostic System to the Determination of Allergen-specific IgE. *Clin Chem*. 1984 Sep;30(9)1467-72.
322. Miller CS, Prihoda TJ. The Environmental Exposure and Sensitivity Inventory (EESI): a standardized approach for measuring chemical intolerances for research and clinical applications. *Toxicol Ind Health*. 1999 Jun; 15(3-4):370-385.
323. Miller CS. In: Doty R, ed. Chemical Hypersensitivity and Multiple Chemical Hypersensitivity Syndrome. Handbook of Olfaction and Gustation, 2nd edition. New York: Marcel Dekker, Inc.; 2003.
324. Miller CS, Ashford NA. In: Spengler J, Samet J, McCarthy J, eds. Multiple Chemical Intolerance and Indoor Air Quality. Indoor Air Quality Handbook. New York: McGraw-Hill, Inc.; 2000.
325. Miller CS. TILT: A New Class of Diseases. How Exposures to Chemicals are Undermining Our Mental and Physical Health. June 6, 2013.
326. Miller CS. LEED: A Set-up for Sick Buildings? Is LEED Diamond the Answer? 2012.
327. McMahon S, Kundomal KA, Yangalasetty S. Pediatric Norms for Visual Contrast Sensitivity Using an APT VCS Tester. *Med Res Arch*. 2017 May;5(5):1-9.
328. Gray MR, Thrasher JD, Hooper D, Dumanov MJ, Cravens H, Jones T. Sphenoid Aspergilloma Diagnosed as a Malignancy: A Case Report. *Otolaryngology*. 2015;5(3)8 pages.
329. Dennis D, Robertson D, Curtis L, Black J. Fungal Exposure Endocrinopathy in Sinusitis with Growth Hormone Deficiency: Dennis-Robertson Syndrome. *Toxicol Ind Health*. 2009 Oct;25(9-10):669–680. https://doi.org/10.1177/0748233709348266.
330. Reinagel M. The Inflammation Free Diet Plan. Published by McGraw-Hill Education. May 21, 2007.
331. Valenzano DR, Terzibasi E, Genade T, Cattaneo A, Domenici L, Cellerino A. Resveratrol Prolongs Lifespan and Retards the Onset of Age-Related Markers in a Short-Lived Vertebrate. *Curr Biol*. 2006 Feb 7;16(3):296-300. https://doi.org/10.1016/j.cub.2005.12.038.
332. Yu X, Li G. Effects of Resveratrol on Longevity, Cognitive Ability and Aging-Related Histological Markers in the Annual Fish Nothobranchius guentheri. *Experimental Gerontol*. 2012 Dec;47(12):940-949. https://doi.org/10.1016/j.exger.2012.08.009.
333. Schwager J, Richard N, Widmer F, Raederstorff D. Resveratrol Distinctively Modulates the Inflammatory Profiles of Immune and Endothelial Cells. *BMC Complement Altern Med*. 2017 Jun 13;17(1):309. https://doi.org/10.1186/s12906-017-1823-z.
334. Rivera L, Moron R, Zarzuelo A, Galisteo M. Long-Term Resveratrol Administration Reduces Metabolic Disturbances and Lowers Blood Pressure in Obese Zucker Rats. *Biochem Pharmacol*. 2009 Mar 15;77(6):1053-1063.
335. Tabrizian K, Shahraki J, Bazzi M, Rezaee R, Jahantigh H, Hashemzaei M. Neuro-Protective Effects of Resveratrol on Carbon Monoxide-Induced Toxicity in Male Rats. *Phytotherapy Res*. 2017 Sep;31(9)1310-1315. https://doi.org/10.1002/ptr.5855.
336. Dennis DP, Thrasher JD. Nasal Fungal Pathology and Trichothecenes Associated with Water-Damaged School and Home. *Austin Journal of Otolaryngology*. 2016;3(1):1-6

337. Pall M. Explaining Unexplained Illnesses: Disease Paradigm for Chronic Fatigue Syndrome, Multiple Chemical Sensitivity, Fibromyalgia, Post-Traumatic Stress Disorder, and Gulf War Syndrome. Published by CRC Press. April 25, 2007.
338. Brewer JH, Thrasher JD, Hooper D. Chronic Illness Associated with Mold and Mycotoxins: Is Naso-Sinus Fungal Biofilm the Culprit? *Toxins*. 2013 Dec 24;6(1):66-80. doi:10.3390/toxins6010066.
339. Hooper DG, Bolton VE, Guildford FT, Straus DC. Mycotoxin Detection in Human Samples from Patients Exposed to Environmental Molds. *Int J Mol Sci.* 2009;10(4):1465-1475. https://doi.org/10.3390/ijms10041465.
340. Grant IH, Thrasher JD, Geller J. Clinical Findings after Indoor Micro-Fungal and Trichothecene Exposure. *Clin Microbiol*. 2017. doi:10.4172/2327-5073.C1.027.
341. Grant IH. Family of 7 Simultaneously Exposed to Extremely High Indoor Trichothecenes and Multiple Toxin-Producing Microfungi: Analysis of Exposure Variables, Clinical Risks for Toxic Exposure Fungal Complications and Clinical Outcomes. *Mycoses*. 2017;60:151-152.
342. Grant IH, Dehmer G, Minamoto GY. Acute Myasthenia Gravis Following Extreme Indoor Hazardous Environmental Exposure: Gliotoxin and Trichothecenes Documented in Thyoma and Superior Efficacy of Antifungal Therapy with Posaconazole: A Case Report." *Mycoses*. 2017;60:149-151.
343. Singer R. Neurotoxicity Guidebook. Published by Aventine Press. 1990.
344. Singer R. Neurotoxicity in Neuropsychology. The Little Black Book of Neuropsychology. 2011;813-838. https://doi.org/10.1007/978-0-387-76978-3_27.
345. Singer R, Johnson DD. Recognizing Neurotoxicity. *TRIAL*. 2006;42(3):62.
346. Petrison L, Johnson E. A Beginner's Guide to Mold Avoidance: Techniques Used by Hundreds of Chronic Multisystem Illness Sufferers to Improve their Health. Published by Paradigm Change. May 29, 2015.
347. Johnson E. Erik on Avoidance: Writings about Mold Avoidance (200-2015). Published by Paradigm Change. September 21, 2015.
348. Thrasher JD. Dr. Jack Dwayne Thrasher, toxicologist. https://drthrasher.org/testing.
349. U.S. Environmental Protection Agency (EPA). Mold Remediation in Schools and Commercial Buildings. EPA 401-K-01-001. Published September 2008. Last updated January 31, 2018. https://www.epa.gov/mold/mold-remediation-schools-and-commercial-buildings-guide.
350. Brasel TL, Martin JM, Carriker CG, Wilson SC, Straus DC. Detection of Airborne *Stachybotrys chartarum* Macrocyclic Trichothecene Mycotoxins in the Indoor Environment. *Appl Environ Microbiol*. 2005 Nov;71(11)7376-7388. doi:10.1128/AEM.71.11.7376-7388.2005.
351. Niemeier, RT, Sivasubramani SK, Reponen T, Grinshpun, SA. Assessment of Fungal Contamination in Moldy Homes: Comparison of Different Methods. *J Occup Environ Hyg*. 2006 May;3(5):262-273. https://doi.org/10.1080/15459620600637333.
352. Johanning E, Gareis M, Nielsen K, Deitrich R, Martlbauer E. Airborne Mycotoxin Sampling and Screening Analysis. Proceedings: *Indoor Air* 2002.
353. Wilson SC, Holder WH, Easterwood KV, Hubbard GD, Johnson RF, Cooley JD, Straus DC. Identification, Remediation, and Monitoring Processes Used in a Mold-Contaminated High School. *Adv Appl Microbiol*. 2004;55:409-423. doi:10.1016/S0065-2164(04)55016-5.
354. U.S. Environmental Protection Agency (EPA). Mold Testing or Sampling. https://www.epa.gov/mold/mold-testing-or-sampling. Page last updated February 22, 2017.
355. U.S. Centers for Disease Control and Prevention (CDC). Basic Facts about Molds in the Environment. https://www.cdc.gov/mold/faqs.htm. Page last reviewed by CDC on December 20, 2017.
356. Brandys R, Brandys G. International Indoor Air Quality Standards and Guidelines for Over 2,000 Chemical and Biological Substances. Published by Occupational & Environmental Health Consulting Services (OEHCS). 2010.
357. Brandys R, Brandys GM. Worldwide Exposure Standards for Mold and Bacteria: With Assessment Guidelines for Air, Water, Dust, Bulk Materials, Insulation, Ductwork and Carpeting. Published by OEHCS Publications. 10th Edition. 2005.
358. New York City Department of Health and Mental Hygiene. Guidelines on Assessment and Remediation of Fungi in Indoor Environments. November 2008. https://www1.nyc.gov/assets/doh/downloads/pdf/epi/epi-mold-guidelines.pdf.
359. Occupational Safety & Health Administration (OSHA). A Brief Guide to Mold in the Workplace, Safety and Health Information Bulletin. SHIB 03-10-10. Last updated 11-08-13. https://www.osha.gov/dts/shib/shib101003.html

360. U.S. Centers for Disease Control and Prevention (CDC). Maintaining Indoor Environmental Quality (IEQ) During Construction and Renovation. Page last updated June 18, 2013. https://www.cdc.gov/niosh/topics/indoorenv/constructionieq.html.
361. Peitzsch M, Bloom E, Haase R, Must A, Larsson L. Remediation of Mould Damaged Building Materials—Efficiency of a Broad Spectrum of Treatments. *J Environ Monit*. 2012(3). doi:10.1039/c2em10806b.
362. Rautiala S, Torvinen E, Torkko P, et al. Potentially Pathogenic, Slow-Growing *Mycobacteria* Released into Workplace Air during Remediation of Buildings. *J Occ Environ Hygiene.* 2004;1:1-16. https://doi.org/10.1080/15459620490250008.
363. Institute of Inspection, Cleaning and Restoration Certification (IICRC). ANSI/IICRC S520 Standard for Professional Mold Remediation, Third Edition. December 8, 2015.
364. Brasel TL, Campbell, AW, Demers RE, Ferguson BS, Fink J, Vojdani A, Wilson SC, Straus DC. Detection of Trichothecene Mycotoxins in Sera from Individuals Exposed to *Stachybotrys chartarum* in Indoor Environments. *Arch Environ Health*. 2003;50(6):317-323. https://doi.org/10.3200/AEOH.58.6.317-323.
365. Occupational Safety & Health Administration (OSHA). Building Assessment, Restoration, and Demolition (Mold Remediation. https://www.osha.gov/SLTC/etools/hurricane/mold.html.
366. U.S. Environmental Protection Agency (EPA). Mold Course Chapter 6: Containment and Personal Protective Equipment (PPE). Last updated on February 21, 2017. https://www.epa.gov/mold/mold-course-chapter-6#Chapter6Lesson4.
367. U.S. Centers for Disease Control & Prevention (CDC). Homeowner's and Renter's Guide to Mold Cleanup after Disasters. Page last reviewed by CDC on September 16, 2017. https://www.cdc.gov/mold/cleanup-guide.html.
368. American Industrial Hygiene Association (AIHA). Recognition, Evaluation and Control of Indoor Mold. 2008.
369. American Industrial Hygiene Association (AIHA): Assessment, Remediation and Post-Remediation Verification of Mold in Buildings. 2004.
370. Wang, J, Fitzpatrick DW, Wilson JR. Effect of T-2 Toxin on Blood-Brain Barrier Permeability Monoamine Oxidase Activity and Protein Synthesis in Rats. *Food Cheml Toxicol*. 1998;36(11):955-961. https://doi.org/10.1016/S0278-6915(98)00079-9.
371. Kebir H, Kreymborg K, Ifergan I, et al. Human TH17 Lymphocytes Promote Blood-Brain Barrier Disruptions and Central Nervous System Inflammation. *Nat Med*. 2007 Oct;13(10):1173-5.
372. Allergy Consumer Review. Safe Room or Haven. February 10, 2003. https://www.allergyconsumerreview.com/safe-rooms.html#sthash.QcSpJyfQ.dpbs.
373. Krouse JH, Derebery J, Chadwick SJ. Managing the Allergic Patient. Published by Elsevier Inc. 2008. https://doi.org/10.1016/B978-1-4160-3677-7.X5001-8.
374. Zhijian L, Zunqiang Z, Yexuan Z, Wei X, Hao L. Investigation of Dust Loading and Culturable Microorganisms of HVAC Systems in 24 Office Buildings in Beijing. *Energy and Buildings*. 2015 Sep 15;103:166-174. https://doi.org/10.1016/j.enbuild.2015.06.056.
375. Biyeyeme Bi Mve, MJ, Cloutier Y, Lacombe N, et al. Comparison of Methods to Evaluate the Fungal Biomass in Heating, Ventilation, and Air-Conditioning (HVAC) Dust. *Environ Monit Assess*. 2017; 189:8. https://doi.org/10.1007/s10661-016-5682-8.
376. Došen I, Andersen B, Phippen CB, Clausen G, Nielsen KF. *Stachybotrys* Mycotoxins: From Culture Extracts to Dust Samples. *Anal Bioanal Chem*. 2016 Aug;408(20):5513-5526. doi:10.1007/s00216-016-9649-y.
377. U.S. Environmental Protection Agency (EPA). Should You Have the Air Ducts in Your Home Cleaned? EPA 402-K-97-002. October 1997. Page last updated on April 12, 2017. https://www.epa.gov/indoor-air-quality-iaq/should-you-have-air-ducts-your-home-cleaned.
378. National Air Duct Cleaners Association (NADCA). Frequently Asked Questions. n.d. https://nadca.com/homeowners/frequently-asked-questions#antimicrobial.
379. Singer R. Neurobehavioral Evaluation of Residual Effects of Acute Chlorine Ingestion. *The Handbook of Forensic Neurotoxicology*. Published by Springer Publishing Company. 2000;54(1):181.
380. Hoffman DD. Bleach Does Not Kill Mold. National Organization of Remediators and Mold Inspectors (NORMI). June 2017. http://www.normi.org/articles/bleach-mold.php.
381. Medina-Ramon M, Zock JP, Kogevinas M, et al. Asthma, Chronic Bronchitis, and Exposure to Irritant Agents in Occupational Domestic Cleaning: A Nested Case-Control Study. *Occ Environ Med*. 2005 Sept;62(9):598-606.
382. Hong S, Kwon H-J, Chio W-J, et al. Association between Exposure to Antimicrobial Household Products and Allergic Symptoms. *Environ Health Toxicol*. 2014 Nov 21;29:e2014017. doi:10.5620/eht.e2014017.

383. Casas L, Espinosa A, et al. Domestic Use of Bleach and Infections in Children: A Multicentre Cross-Sectional Study. *Occup Environ Med*. 2015 Aug;72(8):602-604. doi:10.1136/oemed-2014-102701.
384. U.S. Centers for Disease Control and Prevention (CDC). National Institute for Occupational Safety and Health (NIOSH). International Chemical Safety Card 1119 for Sodium Hypochlorite (chlorine bleach, under the trade name Clorox and others). Page last reviewed by CDC on July 22, 2015. https://www.cdc.gov/niosh/ipcsneng/neng1119.html.
385. Gul S, Savsar A, Tayfa Z. Cytotoxic and Genotoxic Effects of Sodium Hypochlorite on Human Peripheral Lymphocytes in Vitro. *Cytotechnology*. 2009 Mar;59(2):113-119. https://doi.org/10.1007/s10616-009-9201-4.
386. Dangers of Bleach. Educating Wellness. http://www.educatingwellness.com/natural-health/dangers-of-bleach/.
387. National Air Duct Cleaners Association (NADCA). NADCA Position Paper on Chemical Product Applications in HVAC Systems. 2016. https://nadca.com/sites/default/files/docs/2016/chemical_products_position_paper_webfinal.pdf.
388. U.S. Environmental Protection Agency (EPA). A Brief Guide to Mold, Moisture and Your Home. Cleanup and Biocides. EPA 402-K-02-003. Last updated October 19, 2017. https://www.epa.gov/mold/brief-guide-mold-moisture-and-your-home.
389. U.S. Centers for Disease Control and Prevention (CDC). Facts about Chlorine. Page last reviewed by CDC on April 4, 2018. https://emergency.cdc.gov/agent/chlorine/basics/facts.asp.
390. U.S. Centers for Disease Control and Prevention (CDC). Recommendations for the Cleaning and Remediation of Flood-Contaminated HVAC Systems: A Guide for Building Owners and Managers. Page last reviewed by CDC on March 28, 2018. https://www.cdc.gov/niosh/topics/emres/cleaning-flood-hvac.html.
391. Fabry A. Is Your House Making You Sick? A Beginner's Guide to Toxic Mold. Published by CreateSpace Independent Publishing. March 1, 2016.
392. Aleksic B, Draghi M, Sebastien R, et al. Aerosolization of Mycotoxins after Growth of Toxigenic Fungi on Wallpaper. *Appl Environ Microbiol*. 2017 Jun 23;83:e01001-17. https://doi.org/10.1128/AEM.01001-17.
393. Madsen, AM. Effects of Airflow and Changing Humidity on the Aerosolization of Respirable Fungal Fragments and Conidia of *Botrytis cinerea*. *Appl Environ Microbiol*. June 2012;78(11):3999-4007. doi:10.1128/AEM.07879-11.
394. Rautiala S, et al. Potentially Pathogenic, Slow-Growing *Bacillus* Released into Workplace Air during the Remediation of Buildings. *J Occ Environ Hygiene*. 2004;1(1):1-6. https://doi.org/10.1080/15459620490250008..
395. Johanning E, Gareis M, Nielsen K, Dietrich R, Martlbauer. Airborne Mycotoxin Sampling and Screening Analysis. *Indoor Air*. 2002 Jun.
396. Cho SJ, Park JH, Kreiss K, Cox-Ganser JM. Levels of Microbial Agents in Floor Dust during Remediation of a Water-Damaged Office Building. *Indoor Air*. 2011;21(5):417-426. https://doi.org/10.1111/j.1600-0668.2011.00722.x.
397. Singh U, Reponen T, Cho KJ. Airborne Endotoxin and β-D-glucan in PM1 in Agricultural and Home Environments. *Aerosol Air Quality Res*. 2011;11:376-386. doi:10.4209/aaqr.2010.03.0019/
398. Adhikari A, Reponen T, Rylander R. Airborne Fungal Cell Fragments in Homes in Relation to Total Fungal Biomass. *Indoor Air*. 2013;23:142-147. doi:10.1111/j.1600-0668.2012.00799.x.
399. Genna vs Jackson et al. Michigan Court of Appeals, 286 Mich. App. 413; 781 N.W.2d; 2009 Mihc. App. LEXIS 2554. Decided December 15, 2009.
400. Aziz NM, Vasey FB, Leaverton PE, et al. Comparison of Clinical Status among Women Retaining or Removing Gel Breast Implants. *Am J Epidemiol*. 1997;145(11):191.
401. U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID). Medical Management of Biological Casualties Handbook, Seventh Edition. September 2011.
402. U.S. Army, Medical Department of the Army. Medical Aspects of Chemical and Biological Warfare. Textbook of Military Medicine. Published by the Office of the Surgeon General, Department of the Army, United States of America. 1997.
403. Creasia DA, Thurman JD, Jones LJ, et al. Acute Inhalation Toxicity of T-2 Mycotoxin in Mice. *Fundam Appl Toxicol*. 1987 Feb;8(2):230-235. [U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID)].

404. Creasia DA, Thurman JD, Wannemacher RW, Bunner DL. Acute Inhalation Toxicity of T-2 Mycotoxin in the Rat and Guinea Pig. *Fundam Appl Toxicol*. 1990 Jan;14(1):54-59. [U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID)].
405. Pang VF, Lambert RJ, Felsburg PJ, Beasley VR, Buc WB, Haschek WM. Experimental T-2 Toxicosis in Swine Following Inhalation Exposure: Effects on Pulmonary and Systemic Immunity, and Morphologic Changes. *Toxicol Path*. 1987:15(3):308-319. doi:10.1177/019262338701500309.
406. Ochiai E, Kamei K, Watanabe A, et al. Inhalation of *Stachybotrys chartarum* Causes Pulmonary Arterial Hypertension in Mice. *Int J Exp Pathol*. 2008 Jun;89(3):201-208. doi:10.1111/j.1365-2613.2008.00585.x.
407. Kakde UB. Mycotoxins and Its Impact on Human Populations. *MOJ Bioequivalence & Bioavailability*. 2017;3(5).
408. U.S. Government Accountability Office (GAO). Report to the Chairman: Committee on Health, Education, Labor and Pensions, U.S. Senate. Indoor Mold: Better Coordination of Research on Health Effects and More Consistent Guidance Would Improve Federal Efforts. September 2008. https://www.gao.gov/new.items/d08980.pdf.
409. Granirer vs Bakery, Inc. New York Supreme Court. 2014 NY Slip Op 50735(U). Decided April 28, 2014.
410. Gorman vs Crenshaw Lumber et al. Family Settles for $22 Million over Moldy House; Moldy Wood Said to Have Caused Brain Damage in Child. ABC News. November 8, 2005.
411. Gage vs Philadelphia Housing Authority et al. Philadelphia Housing Authority, Others Settle Mold-Related Claims for $11.78 Million. LexisNexis online. May 5, 2010.