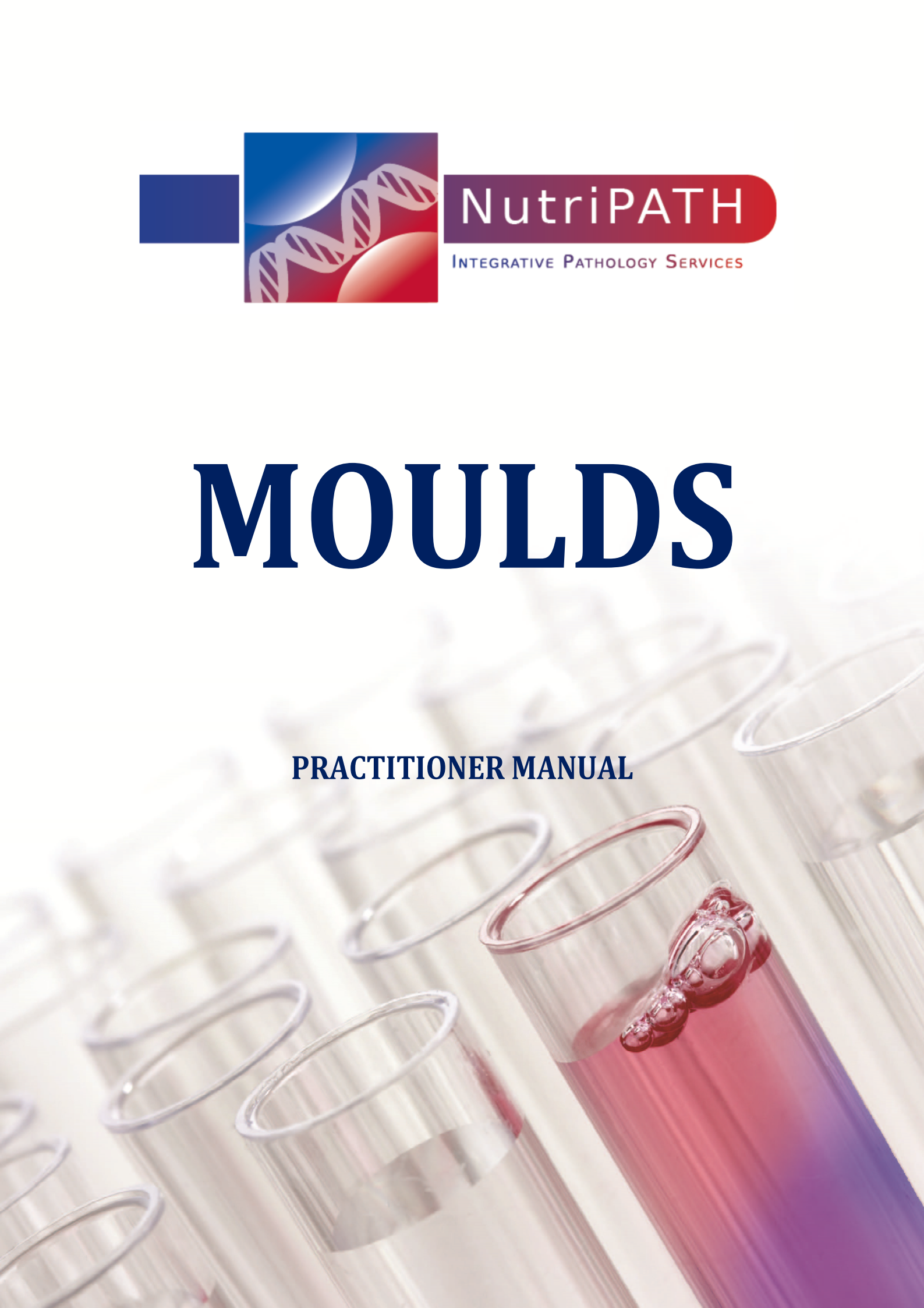
NutriPATH

INTEGRATIVE PATHOLOGY SERVICES

MOULDS

PRACTITIONER MANUAL



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Contents

MOULD ILLNESS

Mould illness is a common but under recognized cause of many debilitating symptoms. As practitioners you may commonly see many people with a list of symptoms. These patients have usually spent much money and time seeing many other physicians for a disparate group of symptoms including chronic fatigue, generalised pain, headaches, depression, sinusitis, rashes, and anxiety. One environmental agent that can cause all of these problems, and more, that never seems to be considered in sunny areas, is mould.

Moulds & Mycotoxins

Many health effects are caused by exposure to the interior environment of water damaged buildings (WDB). The complex mixtures of contaminants present in the air and in the dust in WDB form a toxic chemical stew.

There are so many possible sources of these toxic compounds found in WDB that can lead to the variety of symptoms caused by mould illness, not a single compound can be identified as the sole cause of the inflammatory responses, or the illness, seen in affected patients. Since no one element can be deemed as solely responsible for the sickness, the sole cause becomes the WDB itself.

Below is a list of some of these dangerous compounds and an explanation of each. Please understand this toxic chemical stew is a very complex mixture that truly wreaks havoc in the body.

- **Fungi** - Single-celled or multicellular organisms can be true pathogens that may cause inflammation in healthy persons or they can be opportunistic pathogens that may cause infections and inflammation in immuno-compromised persons.
- **Bacteria** - Single-celled microorganisms which can exist either as independent organisms or as parasites.
- **Actinomycetes** - A group of gram-positive bacteria (order Actinomycetales) that produce various bioactive agents.
- **Mycobacteria** - A large family of bacteria that have unusually waxy cell walls that are resistant to digestion.

- **Mould** - Mould refers to multiple types of fungi that grow in filaments and reproduce by forming spores. Mould may grow indoors or outdoors and thrives in damp, warm, and humid environments. Mould can be found in essentially any environment or season. The most common types of mould found indoors include Cladosporium, Penicillium, Alternaria, and Aspergillus. Stachybotrys chartarum (sometimes referred to as 'black mould') is a greenish-black mould that can be found indoors. It grows on household surfaces with high cellulose content such as wood, fibreboard, gypsum board, paper, dust and lint.

- **Spores** - Not visible to the naked eye produced by mould. Mould spores are very hardy and can survive under conditions in which mould cannot grow, such as in dry and harsh environments. These spores travel through outdoor and indoor air. When mould spores land on a surface where moisture is present, mould can start to grow.
- **Mycotoxins** - toxic chemicals that are present on spores and small fragments of mould and fungus that are released into the air and dust.
- **Endotoxins** - also called lipopolysaccharides (LPS), are cell wall components of gram negative bacteria. They are shed into the environment of WDB upon death of the bacteria. LPS cause inflammatory responses via signalling pathways in the body, releasing inflammatory cytokines. LPS aggravate existing lung disease e.g. asthma, can cause inflammation of the lungs and are synergistic with mycotoxins.
- **Inflammagens** - elicits both oedema and the cellular response of inflammation.
- **Beta glucans** - polysaccharides of D-glucose monomers linked by β-glycosidic bonds.
- **Haemolysins** - exotoxins produced by bacteria that cause lysis of red blood cells in vitro.
- **Microbial Volatile Organic Compounds** (VOCs) - microbes can release organic compounds into the air when there is adequate food supply for such 'secondary metabolite' production. These volatile compounds, called mVOCs for short, can give basements their distinctive musty odour as well as activate innate immune responses in susceptible patients. While we think of fungi as the most common producers of mVOCs, bacteria and actinomycetes are indoor-producers as well.

> "Toxicological evidence obtained in vivo and in vitro supports these findings, showing the occurrence of diverse inflammatory and toxic responses after exposure to microorganisms isolated from damp buildings, including their spores, metabolites and components."
>
> *World Health Organization*

MOULD/BIOTOXIN SYMPTOMS

Mould biotoxins from Stachybotrys, Aspergillus and Fusarium can cause many disparate symptoms and are often misdiagnosed. Dr. Ritchie Shoemaker, a physician from the Eastern Shore in Maryland, has been researching mould and algae biotoxins for the last 15 years. He states the following symptoms can be consistent with mould illness:

- Breathing: Difficult, tightness in chest, shortness of breath on exertion, Asthma
- Diagnoses: Chronic Fatigue, Multiple Chemical Sensitivity, Fibromyalgia, Lyme Disease, Lupus, MS, autoimmune conditions
- Emotions: Irritable, anger, moody
- Extremities: Tingling hands and feet, 'electric shocks', stiff joints in morning
- Eyes: Red, light sensitivity, bloodshot eyes, loss of vision, tearing
- Fatigue: Chronic Fatigue (estimated up to $^{1}/_{3}$ of chronic fatigue), post exertional malaise
- Mental: Confusion, absent mindedness, losing things, brain fog, ADD/ADHD, learning disabilities, anxiety, depression, poor memory, poor word assimilation
- Mould sensitivity: Exposure to damp house, mould in air ducts, reacts to mould
- Nasal: Congestion, nasal soreness, sinusitis.
- Pain: Sudden headaches, sudden, sharp, icepick like
- Skin: Rashes, sensitivity
- Stomach: Cramps, nausea, diarrhoea
- Taste: Metallic
- Thirst: Sense of dryness, excessive thirst, excessive urination
- Weight gain: Sudden, inability to lose weight despite stringent dieting and exercise

This illness affects multiple systems in the body, which causes the patient to exhibit multiple symptoms.

From this list of symptoms it is easy to understand how mould inflammation is commonly misdiagnosed. The symptoms are vague and multiple, since toxins can affect any body organ. Making matters worse is that only 25% of the population seems to be sensitive to mould biotoxins. These 25% seem to be unable to clear mould toxins easily and efficiently. Therefore while many people may be exposed to mould in water damaged home or office building, only 25% will actually get really ill.

Frequently for the ones who get ill it is considered that their symptoms are deemed psychological in nature. However Dr. Shoemaker has documented that the symptoms are real and damaging. In the most severe cases of exposure multiple sclerosis, blindness and even death can result. A ground-breaking public health study, based on data from 5,882 adults in 2,982 households, has found a solid connection between damp, mouldy homes and depression. (Stachybotrys chartarum, trichothecene mycotoxins, and damp building-related illness: new insights into a public health enigma. Pestka JJ, Yike I, Dearborn DG, Ward MD, Harkema JR Toxicol Sci. 2008;104(1):4.)

Suspect Mould

The first step to diagnosing mould illness is to suspect it. However most people often see one doctor for their skin issues, another for their sinusitis and a third for their anxiety. No one puts it together that all the symptoms are being caused by mould, and the patient receives at least three different prescriptions none of which solve the problem. Anyone living in a house with mould, who is suffering from some of the previously mentioned symptoms, should consider getting more testing. Since mould toxins cause inflammation through activation of cytokines, there are also a number of lab tests which can show the effects of inflammation. However none are definitive by themselves for the presence of mycotoxins.

Buildings can host fungi, bacteria, mycobacteria, and actinomycetes as a result of construction defects like inappropriate ventilation, faulty construction of crawl spaces, inadequate building design, flat roofs, fake stucco cladding without adequate caulking, basements exposed to saturated ground water conditions, not correcting water leaks and more.

An article of interest: **www.survivingmold.com/community/mary-ackerley-the-brain-on-fire-the-role-of-toxic-mold-in-triggering-psychiatric-symptoms**

Dr Ritchie Shoemaker, M. D.

Ritchie Shoemaker, M. D., is a recognised leader in patient care, research and education pioneer in the field of biotoxin related illness. While illness acquired following exposure to the interior environment of water-damaged buildings (WDB) comprised the bulk of Shoemaker's daily practice, other illnesses caused by exposure to biologically produced toxins are quite similar in their "final common pathway." What this means is that while the illness might begin acutely with exposure to fungi, spirochetes, apicomplexans, dinoflagellates and cyanobacteria, for example, in its chronic form, each of these illnesses has similar symptoms, lab findings and Visual Contrast Sensitivity findings. Taken together the inflammatory illness from each of these diverse sources is known as a **Chronic Inflammatory Response Syndrome (CIRS)**.

> The definition of CIRS is "an acute and chronic, systemic inflammatory response syndrome acquired following exposure to the interior environment of a water-damaged building with resident toxigenic organisms, including, but not limited to, fungi, bacteria, actinomycetes and mycobacteria as well as inflammagens such as endotoxins, betaglucans, haemolysins, proteases, mannans as well as volatile organic compounds".

Dr Shoemaker says, "I suspect that the next textbook of autoimmunity and rheumatology will be one dedicated to treating high TGFβ1 and restoring control of T-regulatory cells. Similarly, no one will be seen for neurological deficits and pulmonary problems without consideration of nerves and lungs as targets of innate immune responses gone haywire. As it is now we see unusual cases of multiple sclerosis, idiopathic juvenile arthritis, interstitial lung disease and many others unveiled as treatable conditions where the therapeutic target is lack of regulation of innate immune inflammation."

Since 1998, Dr. Shoemaker has treated over 7,000 patients with an illness caused by exposure to these conditions. Each patient has a syndrome that is readily identified by blood tests performed in standard medical labs all over the country. These illnesses reflect a growing societal problem: dangerous buildings. Inhaling these dangerous inflammagens makes people sick.

See more at his website www.survivingmold.com

THE BIOTOXIN PATHWAY

Stage 1: Biotoxin Effects

It all starts when a person is exposed to a biotoxin. In most people, the biotoxin is 'tagged' and identified by the body's immune system and is broken down and removed from the blood by the liver. However, some individuals do not have the immune response genes that are required to eventually form an antibody to a given foreign antigen. In these cases the biotoxins are not 'tagged' and remain in the body indefinitely, free to circulate and create inflammation.

Once present in the body, the biotoxins begin to set off a complex cascade of biochemical events. The biotoxin binds to surface receptors (Toll receptors and many more) in nearly every cell in the body. This recognition and binding of the biotoxin causes a continual upregulation of multiple inflammatory pathways, including production of cytokines, split product of complement, and TGFβ1. Biotoxins also directly affect nerve cell function, which is one of the reasons that the symptoms are useful in diagnosis.

Stage 2: Cytokine Effects

Cytokines in turn bind to their receptors, causing release of MMP-9 in blood. In the brain, cytokines bind to the leptin receptor, preventing its normal function in the hypothalamus. The blocked leptin receptor will no longer create the initiation of steps that lead to production of melanocyte stimulating hormone (MSH). Elevated cytokines can produce many different symptoms including: headache, muscle ache, unstable temperature and difficulty concentrating. This problem is due to MSH deficiency. Of importance in cardiovascular health, MMP-9 delivers inflammatory elements from the blood into sensitive tissues and can combine with plasminogen activator inhibitor (PAI-1) to increase clot formation and arterial blockage.

Stage 3: Reduced VEGF

The elevated cytokine levels in the capillaries attract white blood cells, leading to restricted blood flow and lower oxygen levels in the tissues (capillary hypoperfusion). Reduced vascular endothelial growth factor (VEGF) leads to fatigue, muscle cramps and shortness of breath.

Stage 4: Immune System Effects

Patients with certain HLA genotypes (immunity related genes) may develop inappropriate immune responses which may include antibodies to gliadin (gluten sensitivity); cardiolipins (affects blood clotting). Most devastatingly of all, the complement system becomes chronically activated resulting in high levels of C4a.

Stage 5: Low MSH

Reduced MSH production results in yet another set of problems and symptoms. The reduced production of melatonin results in sleep problems. Endorphin production is suppressed which leads to chronic and sometimes unusual pain. Lack of MSH can cause malabsorption or 'leaky gut' which further weakens and deregulates the immune system. White blood cells eventually lose regulation of cytokine response so that opportunistic infections may occur or recovery from infections is slower.

Stage 6: Antibiotic Resistant Staph Bacteria

Reduced MSH also allows multiple antibiotic-resistant coagulase-negative staph (MARCoNS) to survive in biofilm on the mucous membranes. These bacteria further compound MSH deficiency and the problem by producing exotoxins A and B that cleave MSH, further decreasing the MSH levels.

Stage 7: Pituitary Hormone Effects

Reduced MSH can decrease pituitary production of antidiuretic hormone (ADH) which can lead to thirst, frequent urination, neurally-mediated hypotension, low blood volume, and electric shocks from static electricity. While sex hormone production is often down-regulated the pituitary may upregulate the production of cortisol and ACTH in the early stages of illness, and then drop to abnormally low or low-normal ranges later.

SUMMARY OF CHRONIC INFLAMMATORY RESPONSE SYNDROME (CIRS)

- Biotoxins induce inflammatory cytokines in the genetically susceptible including C4a, TGF-β-1, MMP-9.
- These cytokines disrupt regulatory neuropeptides including MSH and VIP.
- Raised TGF-β-1 shifts balance away from T regulatory cells to pathogenic Th17 cells; further aggravating inflammation.
- Hence the progression to a multi-system, multi-symptom illness of CIRS.

Can a patient have two sources of CIRS? Yes and often do.

TREATMENT

The first line of treatment for mould illness is removal from the source. The client is unlikely to get completely well in a house or office building where mould toxins are continually present.

Remediation by a specialist needs to be done in order to keep the problem from getting worse. Since the mould spores may be dispersed through the house like a fine mist it is sometimes necessary to get rid of clothing, linens, furniture and photos. Some people have left their houses and never returned.

The next step in treatment involves pulling the toxins from the body. About 75% of the population has immune systems that are much more efficient than others in doing this.

Since the toxins are eliminated in bile, drugs that are known as bile sequestrants, such as cholestyramine are used. Cholestyramine is a prescription medication.

SHOEMAKER MOULD ILLNESS TREATMENT

There are a series of steps to be taken, each in its proper order.

If the patient feels fine with simple removal from exposure, no more steps are needed, though note there are a few people who are fixed with this step alone.

1. Differential diagnosis – Pathology testing is needed to show inflammatory abnormalities.

2. Performing Environmental Relative Mouldiness Index (ERMI) testing to ensure there is no exposure to a building with an ERMI greater than 2 if the patient's MSH is less than 35 and C4a is less than 20,000; or no exposure to ERMI greater than negative 1 if MSH is less than 35 and C4a is greater than 20,000. www.epa.gov/microbes/moldtech.htm

3. Removal from exposure - This means no more working, schooling or living in a mouldy environment for WDB illness patients.

4. Correcting toxins in the body with cholestyramine (compounded) or as Questran Lite, using VCS monitoring to assess progress monthly.

5. Eradicate biofilm-forming MARCoNS if positive with BEG nasal spray (Bactroban, EDTA & gentamycin/rifampicin) - Bacteria create hemolysins which increase cytokine levels and exotoxins which depress MSH levels.

6. Eliminate gluten if positive anti-gliadin. Rule out coeliac disease.

7. Correct elevated MMP-9 with fish oils and an amylose-free diet.

8. Correct ADH/osmolality if required with desmopressin.

9. Correct low VEGF – Fish oils and amylose-free diet can work here too.

10. Correct elevated C3a with high-dose statins and CoQ10. *NOTE: C3a often elevated in early Lyme but rare to see in chronic pts who have not had recent tick bite.*

11. Correct elevated C4a.

12. Reduce elevated TGF β-1.

13. Replace low VIP with VIP spray – ensure VCS normalised, negative MARCoNS, normal lipase levels and no exposure to WDB for best outcome.
Check androgen levels (DHEA, Testosterone, and Oestrogen) as low VIP can cause elevated aromatase levels.

14. Final checks to verify stability off supplements.

KEY MARKERS IN BIOTOXIN ILLNESS

Biomarker	Physiology	Significance
Elevated TGF β-1	Causes T regulatory cell dysfunction; complement splitting	Increased pathogenic T cell production, immune suppression
Elevated C4a & C3a	Anaphylatoxins attract & activates mast cells & basophils	Increased vascular permeability; mucosal bleeding; capillary hypo perfusion (oedema, reduced VO2 max & high lactate on MRS)
Elevated MMP-9	Collagenase for ECM/tissue barrier proteolysis & remodelling	Neuroimmune injury likely
Elevated autoantibodies e.g. antigliadin abs, cardiolipin abs etc	Gut wall damage; mitochondrial damage	Immune dysregulation raises risk of auto-immunity; linked HLA-DQ genes
Low T regulatory cells	Impaired immune modulation & tolerance to self-antigens	Increased risk of auto-immunity & cancer growth
Low MSH & secondary low ADH	Impaired neuropeptide regulation of immune defense at mucous membranes & lowered pituitary stimulation of sex hormones	Hyperpermeable/leaky barriers; chronic membrane inflammation; thirst, increased urination, POTS (ppostural orthostatic tachycardia syndrome), electric shocks
Elevated PAI-1	Protease inhibitor, positive regulation IL-8 + leukotrienes + angiogenesis + response to LPS + neg regulation apoptosis	Combines with MMP-9 to increase clot formation & arterial blockage
Low VEGF	Chronic tissue hypoxia & impaired response to capillary hypoperfusion	Symptoms of fatigue, SOB, muscle cramps. Can treat with erythropoietin.
Low VIP	Impaired vasodilation; downregulation of Th1/Th2 response	Poorly controlled inflammation & reduced microcirculatory blood flow
Elevated Leptin	Leptin resistance and reduced signalling	Impaired satiety & obesity risk
Low ACTH	Reduced adrenal cortisol production	Chronic inflammation & impaired stress resilience

REPORT COMMENTS

Leptin comment:

Damaged Leptin receptors in the hypothalamus, caused by elevated cytokines, leads to reduced production of MSH.

Leptin turns on how tightly the body holds onto fatty acids. When Leptin is high, one holds onto fatty acids and stores them in fat. This leads to rapid weight gain, and because of the high Leptin, standard approaches to weight loss like eating less and exercising more will fail, as the body is non responsive to exercise and diet. The inflammatory responses that cause Leptin levels to rise lead to patients who are chronically tired, in chronic pain, and forever overweight.

Vasoactive Intestinal Polypeptide (VIP) comment:

Vasoactive Intestinal Polypeptide (VIP) is a neuroregulatory hormone with receptors in the hypothalamus. This hormone/cytokine regulates peripheral cytokine responses, pulmonary artery pressures, and inflammatory responses throughout the body.

Low VIP levels are present in mould illness patients. This leads to unusual shortness of breath, especially in exercise. VIP plays a role similar to MSH in regulating inflammatory responses.

With respect to the digestive system, VIP seems to induce smooth muscle relaxation (lower oesophageal sphincter, stomach, gall bladder), stimulate secretion of water into pancreatic juice and bile, and cause inhibition of gastric acid secretion and absorption from the intestinal lumen, which can lead to chronic, watery diarrhoea.

Matrix metallopeptidase 9 (MMP-9) comment:

Matrix metallopeptidase 9 (MMP-9) is an enzyme involved in the breakdown of extracellular matrix in normal physiological processes, such as embryonic development, reproduction, and tissue remodelling, as well as in disease processes.

It has been implicated in pathogenesis COPD by destruction of lung elastin, in rheumatoid arthritis, atherosclerosis, cardiomyopathy, and abdominal aortic aneurysm.

MMP-9 delivers inflammatory elements of blood into subintimal spaces, where further delivery into solid organs (brain, lung, muscle, peripheral nerve and joint) is initiated.

Elevated cytokines levels generate flu-like symptoms (Headache, muscle aches, fatigue, unstable body temperature, difficulty concentrating.

Elevated cytokines also lead to elevated MMP-9 levels which increase the delivery of inflammatory elements from the bloodstream to the brain, lungs, joints, thus increasing clot formation and arterial blockage.

Vascular Endothelial Growth Factor (VEGF) comment:

Vascular endothelial growth factor (VEGF) is a substance made by cells that stimulates new blood vessel formation and increases blood flow in the capillary beds. VEGF is a polypeptide.

Deficiency of VEGF is quite common and is a serious problem in biotoxin illness patients that must be corrected. If you don't have blood flow, cells begin starve and don't work properly.

Reduced VEGF levels lead to fatigue/muscle cramps, shortness of breath.

Caused by effects of increased cytokine levels in capillaries leading to restricted blood flow.

TGF Beta-1 (TGFβ1) comment:

TGF β-1 is a protein that has important regulatory effects throughout innate immune pathways. This protein helps control the growth and division (proliferation) of cells, the process by which cells mature to carry out specific functions (differentiation), cell movement (motility), and the self-destruction of cells (apoptosis). The TGF β-1 protein is found throughout the body and plays a role in development before birth, the formation of blood vessels, the regulation of muscle tissue and body fat development, wound healing, and immune system function (especially regulatory T-cells).

TGF β-1 can impair T-regulatory cell function, which in turn contributes to the activation of autoimmunity, yet TGF β-1 also plays a role in suppressing autoimmunity. TGF β-1 has become important in the exploding incidences of childhood asthma, raising the tantalizing issue of remodelling due to biotoxin exposure. The EPA says that 21% of all new cases of asthma are due to exposure to water damaged buildings. If an individual develops wheezing after exposure to a water damaged building, look for remodelling to be the cause. Remodelling means 'something' happens that the airway changes to be more reactive and in need of medications to reduce wheezing. Neurologic, autoimmune and many other systemic problems also are found with high TGF β-1.

TGF β-1 is associated with respiratory problems, particularly asthma-like symptoms, tremors (reminiscent of those associated with Parkinson's disease), as well as scarring on the brain, and symptoms similar to those of multiple sclerosis.

Whenever autoimmunity is identified (elevated TGF β-1 with low CD4+CD25++ cells drives production of antibodies to gliadin and cardiolipin), remove gluten from the diet for a minimum three months.

C4a has become the inflammatory marker of greatest significance looking at innate immune responses in those with exposure to Water Damaged Buildings (WDB).

The complement system is a group of proteins that move freely through your bloodstream. The proteins work with your immune system and play a role in the development of inflammation.

Each complement activates inflammatory responses, with spill over of effect from the innate immune response to acquired immune response and hematologic parameters.

These short-lived products are re-manufactured rapidly, such that an initial rise of plasma levels may be seen within approximately 12 hours of exposure to biotoxins, and sustained elevation is seen until definitive therapy is initiated.

Melanocyte Stimulating Hormone (MSH) comment:

Melanocyte stimulating hormone (MSH) has multiple anti-inflammatory and neurohormonal regulatory functions, exerting regulatory control on peripheral cytokine release, along with both anterior and posterior pituitary function.

In mould illnesses, MSH is generally found to be low in over 95% of patients. This means increased susceptibility to mould illness, ongoing fatigue, pain, hormone abnormalities, mood swings, and much more. As MSH is a hormone, called a regulatory neuropeptide, and it controls many other hormones, inflammation pathways, and basic defences against invading microbes, a lack of MSH can lead to chronic sleep disorders with non-restful sleep develop, and endorphin production is reduced, so chronic pain follows.

Resistance to Staph bacteria - these multi antibiotic resistant bacteria release chemical substances that further aggravate the levels of elevated cytokines and reduced MSH.

As MSH regulates other hormones, especially antidiuretic hormone—you'll see people that are thirsty and urinate more frequently. And they get static shocks.

MSH also has effect on gonadotrophins (oestrogen and testosterone primarily). You'll find abnormalities of androgens in about 40% of patients. Part of the mechanism for these androgen problems is aromatase as it is upregulated.

Unfortunately, many of these people will also present with low testosterone, and their doctors will often times prescribe testosterone as a result. However, if they are biotoxic, aromatase will convert more testosterone into oestrogen, resulting in a worsening of symptoms.

AntiDiuretic Hormone (ADH) comment:

Antidiuretic hormone (ADH), or vasopressin, is a substance produced by the hypothalamus and released by the pituitary gland. The hormone controls the amount of water your body removes.

Osmolality is a test that measures the concentration of all chemical particles found in the fluid part of the blood.

Symptoms associated with dysregulation of ADH include dehydration, frequent urination, with urine showing low specific gravity; excessive thirst and sensitivity to static electrical shocks; as well as oedema and rapid weight gain due to fluid retention during initial correction of ADH deficits.

References & Resources

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