MYCOTOXINS INTERVENTION GUIDE

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What are Mycotoxins?

Mycotoxins are a structurally diverse group of compounds, produced mainly by fungi, or molds, under suitable temperature and humidity conditions. Mycotoxins may develop on various foods and agricultural crops, causing serious risks for human and animal health. Currently, more than 300 mycotoxins are known, but scientific attention is focused mainly on those that have proven to be carcinogenic and/or toxic.

Human exposure to mycotoxins occurs through ingestion of contaminated foods and dirt, through skin contact with contaminated surfaces, and inhalation of air and dust containing mycotoxins.

Food sources of mycotoxins include plant-derived foods that are contaminated with mycotoxins and mycotoxins and their metabolites in animal products such as meat and eggs. Unfortunately, mycotoxins are resistant to most food processing methods, including heat.

In addition, water damaged buildings are a common route of mycotoxin exposure, and one that often goes unrecognized as molds can grow on almost any wet and warm place including inner walls of buildings, fiberglass insulation, and wall paper, among others.

Mycotoxins produced by molds may differ based on the species of mold and how toxigenic the species is. A single mold species may produce several different mycotoxins. Toxic effects can be acute, chronic, mutagenic, or teratogenic.

Harmful Effects of Mycotoxins

Mycotoxin illnesses, called mycotoxicoses or mycoses, can range from mild and reversible (e.g, athlete's foot) to irreversible organ damage and life-threatening (e.g., invasive aspergillosis) depending upon the specific mycotoxin, the health of the individual, exposure levels, time course of exposure, mycotoxin interactions, and other circumstances.

The molds that cause mycoses can be divided into two categories: ***primary pathogens*** that affect healthy people, or ***opportunistic pathogens*** that affect people who are chronically ill or immune-suppressed

Mycotoxins' pathogenic effects are multifactorial and complex and can target all organ systems, including the nervous system, cardiovascular system, respiratory system, digestive system, urinary system, immune system, endocrine system, reproductive system, and integumentary system.

Oxidative stress and inflammation are mechanisms that contribute to mycotoxin-induced toxicity. Mycotoxins have been shown to induce intracellular glutathione depletion, and studies suggest that antioxidants may mitigate the toxic effect of mycotoxins associated with oxidative stress.

Mycotoxins activate an inflammatory response in human macrophages, intestinal inflammatory diseases, airway inflammation, and atopic dermatitis.

Mycotoxins have been shown to impact micronutrient absorption, distribution, metabolism, and excretion in livestock with mycotoxicoses.

For example, aflatoxins decreased absorption of calcium and decreased hydroxylation of vitamin D3 in animal studies. Additionally, while the effects in humans are less clear, animal studies suggest that the immunological suppression associated with mycotoxins may be further exacerbated in cases of zinc, iron, and vitamin A deficiency.

Studies have demonstrated that mycotoxins cause mitochondrial dysfunction by formation of mitochondrial DNA adducts, inhibition of protein synthesis, disruption of cristae, pleomorphism, membrane damage and induction of cell death (apoptosis).

Conventional Medicine Treatments for Mycotoxin Exposure

Immunotherapy and steroids are used to reduce allergic sensitization to mycotoxins. Mycotoxin-related infections are treated with antifungal agents, however antifungal resistance and the presence of biofilms may impact the effectiveness of treatment. Decongestants, antihistamines, and anti-inflammatory agents are also used to treat mycotoxin-related symptoms.

Functional Medicine Treatments for Mycotoxin Exposure

In addition to removal of the initial source of mold and mycotoxin exposure from diet or environment, there are a number of supportive supplemental therapies that can aid in full body detoxification of mycotoxins.

Because mycotoxins are so inflammatory to the liver's normal detoxification capacity, detoxification is often impaired and the individual may become susceptible to multiple toxicities during this time. Supporting healthy liver detoxification, and keeping all detoxification pathways open and functional is critical to elimination of toxins from all sources, to reduce overall toxic load.

Supplements that bind mycotoxins to increase excretion:

Activated Charcoal: Activated charcoal is able to ***adsorb*** toxic substances in the gut, and possibly systemically, therefore is a top choice for mycotoxin binding during treatment. Activated charcoal can, however, bind other supplements, and, therefore, should be taken at least 90 minutes before or after any other supplements taken orally.

Bentonite clay: Bentonite clay is a byproduct of a highly nutritious volcanic ash, which is high in minerals and other charged particles. These positively charged ions are able to bind negatively charged ions and other toxins, including some heavy metals. Bentonite clay usually is safe to use with other supplements and vitamins, and may reduce the need for multi-mineral supplements.

Liver Detoxification and Antioxidant Support:

Glutathione: Glutathione reduces oxidative stress from exposure to mycotoxins and promotes detoxification of a number of toxic substances. It is also the master antioxidant, which gives it the ability to regenerate other antioxidant vitamins, including vitamins A, C, and E.

Supplemental glutathione is typically poorly absorbed because it is denatured during both acidic and enzymatic digestion. When using oral glutathione supplements, a liposomal or enteric-coated form of glutathione may be more effective at raising systemic glutathione levels, particularly intracellular glutathione.

Intravenous (IV) glutathione injections may be even better absorbed and beneficial to patients affected by mold and mycotoxin illnesses, due to being able to bypass gastrointestinal digestion and absorption.

Antioxidant vitamins that should be supported through diet and additional supplementation where appropriate:

- Vitamin A
- Vitamin C
- Vitamin E
- Melatonin
- CoQ10
- Licorice
- Zinc
- Probiotics



Additional Supplemental Therapies:

Nasal passage rinse: This may be beneficial for those affected by mold and mycotoxins, especially with upper respiratory symptoms, to help clear mycotoxins from the nasal passage, which is one of the first places they land in the human body.

Citricidal nasal spray: This can reduce the chance of sinusitis upon first exposure to mold.

Beta glucans: Beta glucans are a type of fiber commonly found in the cell walls of a variety of plants, including mushrooms, baker's yeast, and whole grains. They can also be found in supplement form. These chemical compounds can stimulate the activity of macrophages, which are special immune cells that attack and devour invading pathogens.

Fungal overgrowth concerns:

In some susceptible individuals, Candida albicans and other opportunistic fungi may overgrow and present an additional layer of infection that may need to be addressed. Consider stool testing using the Vibrant Gut Zoomer Complete to assess the presence of Candida, bacterial pathogens, and parasites.



Anti-candida products: In case of candida infection from continued mold exposure, effective antifungal products may include:

- Caprylic acid
- Monolaurin or Lauricidin
- Pau d'arco
- Olive leaf
- Oregano oil
- Garlic
- Berberine
- Undecylenic Acid (Calcium Undecylenate)
- Grape seed extract
- Horopito
- Saccharomyces boulardii
- Aloe vera

Complementary and Supportive Lifestyle Interventions

Sauna therapy: Saunas are a powerful tool to enhance detoxification. Because some toxins are difficult to excrete through renal pathways, and when digestive function is impaired, reducing biliary output of bound toxins, sweating is often the alternative exocrine pathway of detoxification that the body prefers to use.

Saunas can be in dry, steam, or infrared formats and sufficient time should be spent to ensure that sweating occurs on a regular basis. Sauna therapy can be used daily, especially when patients affected by mold and mycotoxins are severe enough to cause fatigue that prevents physical exercise-induced sweating.

Exercise: Exercise can promote movement of lymph system and sweating, both of which encourage detox, boost mood, and the immune system. If a patient feels they are able to perform mild to moderate intensity exercise regularly, this should be encouraged in addition to other lifestyle, nutritional, and supplemental therapies.

Meditation/tapping: A common symptom of mold and mycotoxin exposure is anxiety. Therapies that reduce anxiety include meditation and tapping. Meditation can be either guided or silent. Overall time spent daily meditating can vary by person and does not have to be done in one continuous block of time, but could be done in short sessions throughout the day.

Tapping, also known as emotional freedom technique (EFT), is a therapy in which acupressure points are stimulated to release tension and anxiety, supporting emotional and psychological health.

Diet: Nutrition is a critical piece of healing from mold and mycotoxin illnesses. As the foundation for nutrients that support all organ and immune system function to improve health and detoxification, the quantity and quality of nutrients during treatment cannot be underestimated.

Prioritize dark colored organic fruits and vegetables to enhance overall antioxidant and nutrient intake. Eat plenty of apples, cruciferous vegetables, and food sources of lycopene. Avoid high sugar, processed food (cakes, pies, donuts, pastries, candies, etc), and avoid moldy foods such as peanuts, blue cheese, and fermented foods.

Environmental Considerations for Mold Exposure

Mold remediation should be accomplished while or prior to attempting to treat clinical mycotoxin exposure. If environmental sources of mold are not removed and eliminated, treatment for internal mycotoxin/mold in the affected individual will be unsuccessful.

Mold spores can travel throughout an indoor space and collect in dust and on other surfaces. While removing the water-damaged surfaces is critical to mold remediation, thorough cleaning of all surfaces indoors should be accomplished after remediation, in order to ensure that spores are not able to flourish elsewhere and continue to infect humans in the area.

Air Ducts

After removing water-damaged sources of mold in a building, but before cleaning other surfaces, test the air outflow coming from ventilation to determine if toxic mold spores have infiltrated the ventilation system and are continuing to spread throughout the air in the building. If there is a serious toxic mold infestation anywhere in a building, airborne mold spores from such mold locations will usually enter and contaminate the heating/cooling equipment and ducts. Consider having a professional contractor with experience in mold remediation test for this.

Indoor Air Quality

Consider using industrial strength HEPA filters in fans or other filtered air devices that can remove and isolate mold spores, preventing them from re-entering the circulated air in the building/office/home.

Air purifiers do not all remove very small particles such as mold spores. Only true HEPA filters will do this, so check with the manufacturer of the air purifier you own or are considering purchasing to verify which type and size of particles its filter will remove. Change filters often (preferrably by taking the air purifier outdoors before opening up the filter casing) when using for mold remediation purposes.

Cleaning Surfaces

Before cleaning surfaces that have been exposed to mold or mold spores, wear eye protection and a respirator mask that has been approved for use in water-damaged areas. If a large area has been water-damaged, consider hiring a professional contractor with experience in mold remediation to remove large areas of damaged wall, floor, carpet, or other structures in a building.

After removal of the water-damaged areas is complete, all exposed surfaces in the indoor environment should be wiped down with one or more anti-fungal products to kill and inhibit mold. This includes:

- walls
- baseboards
- crown molding
- windows/window sills
- blinds
- furniture such as (desks, filing cabinets, bookshelves, bed frames,dressers, etc)



- Fabric surfaces should be washed with Borax laundry detergent; if a surface cannot be placed in a washing machine, take the item outside to brush off excess mold so as not to continue to spread spores inside, then consider a fabric-safe anti-fungal spray applied daily for 2-3 weeks; fabric surfaces may include: bedding, curtains/drapes, shower curtains, bath rugs, throw rugs, couch/chair upholstery, throw blankets/pillows, and pet beds

- wipe down tops and bindings of books and papers exposed
- electronics
- clean floor and ceiling fans thoroughly with solution, including interior surfaces and replace filters from tower fans/air purifiers; discard fans or air flow devices that cannot be taken apart and cleaned thoroughly
- replace filters in vacuums
- wash all clothing exposed

Anti-fungal products

There are a number of anti-fungal products available commercially at hardware or home stores designed to aid in cleaning surfaces which have been exposed to mold and mold spores.

Commercial products often contain bleach or ammonia, and may not be recommended for use indoors in poorly ventilated areas. Look for products that contain ingredients such as:



- hydrogen peroxide
- chlorine bleach*
- ammonia
- distilled white vinegar
- baking soda and borax

**some sources consider chlorine bleach to not be an effective toxic mold-killing agent and not effective against total elimination of mold spores*

Anti-fungal essential oils

There are also a number of essential oil extracts that are extremely effective anti-fungal agents, when used in the proper concentration regularly. These extracts are generally mixed in a ratio of 1 tsp oil : 1 cup water, then used in a spray bottle or other method of liquid application.



- Oregano
- Tea Tree
- Clove
- Cinnamon
- Thyme
- Grapefruit seed extract

To make a DIY home remedy of essential oils to clean surfaces in your home or workplace, mix 1 tsp oil of each oil per 3 cups water (3 total tsp of oil):

Tea tree oil *Clove oil* *Cinnamon oil*

Odor will be strong, so use in a well-ventilated area, preferably where windows can be opened to allow air flow and aid in drying.

Using a Nebulizing Diffuser

The incorporation of a nebulizing diffuser may be helpful in post-mold removal building maintenance. A nebulizing diffuser takes essential oils and heats them into a particulate gas-like state, where they can be pushed into the surrounding air, but does not use water, like other diffusers.

It is important to use a diffuser that does not use water when diffusing essential oils after mold removal, so as not to add more moisture to the environment.

Any of the mold killing essential oils can be used in a nebulizing diffuser, depending on what aroma you prefer in the area being treated.

Humidity Control

There are 3 things necessary for mold growth:

Mold spores: there will always be a supply because they are in the air outside and can easily infiltrate your home/building

Porous surfaces for the mold to feed on like wood, wall board, etc.

Moisture with humidity 60% and higher

A critical part of mold remediation in environmental maintenance of buildings to prevent further mold growth is controlling the humidity indoors. Mold cannot grow in dry environments, therefore, using a dehumidifier in affected areas, to reduce humidity to below 60%, can reduce the possibility of mold re-growing.

Humidity levels below 50% will cause current mold to go dormant, but will not completely kill it.

Dehumidifiers come in a variety of sizes, so be sure to check how much square footage the humidifier you have can cover, depending on the size of the area that was water-damaged. Some better quality dehumidifiers also come with air purifiers that can remove mold spores that may still be circulating.





DESCRIPTION OF MYCOTOXINS

Mycotoxins

Mycotoxins are a structurally diverse group of mostly small molecular weight compounds, produced mainly by the secondary metabolism of some filamentous fungi, or molds.

Aflatoxins

Aflatoxins are found mainly on food-derived molds. They are secondary metabolites produced by different strains of Aspergillus species, widely found as contaminants in a great variety of crops—cereals, oilseeds, tree nuts and spices.

Aflatoxin exposure can lead to a greater risk for liver cancer.

Common crops which have been found to contain aflatoxins include: cassava, chili peppers, corn, cotton seed, millet, peanuts, rice, sesame seeds, sorghum, sunflower seeds, tree nuts, wheat, and a variety of spices. When contaminated food is processed, aflatoxins enter the general food supply where they have been found in both pet and human foods, as well as in feedstocks for agricultural animals.

Aflatoxins are most commonly ingested. However the most toxic type of aflatoxin, B1 can absorb through the skin.

Aflatoxin M1

Aflatoxin M1 (AFM1) is the principal hydroxylated aflatoxin metabolite of Aflatoxin B1 (AFB1), the most recurrent and most harmful aflatoxin present in the milk of dairy cows fed a diet contaminated with AFB1. AFB1 can accumulate in animals which consume contaminated grain and feed sources.

Carry-over of AFB1 as AFM1 in the milk of dairy cows has been established to range from 0.3% to 6.2%. Due to the high stability of AFM1 towards milk processing technologies, such as pasteurization, ultra-high temperature heating (UHT), and other processing methods, this mycotoxin can be found not only in milk, but also in dairy products, usually at higher concentration than that found in raw milk. In addition, AFM1 is found in human breast milk too.

This mycotoxin has become a real public health concern, especially for infants and young children. It is considered that infants are more exposed to AFM1 contamination by breast milk intake than those using infant formula.[1] Moreover, the International Agency for Research on Cancer (IARC) classified AFB1 and AFM1 as human carcinogens belonging to Group 1 and Group 2B, respectively.[2]

Aflatoxin B1

Aflatoxin B1 (AFB1) is produced by many strains of Aspergillus fungi. Aflatoxin B1 is the most potent natural carcinogen known and is usually the major aflatoxin produced by toxigenic strains. Aflatoxin B1 is one of the most potent liver carcinogens known and has been associated as a cocarcinogen with hepatitis B in the high incidence of human liver cancer.

Warm temperatures, high humidity, and plant injuries, in the field and during storage, promote both the growth of the fungi and aflatoxin production. The greatest threat to public health is from contaminated peanuts, cottonseed, maize, and rice. AFB1 is a potent toxin, mutagen, and carcinogen, and is implicated in the etiology of hepatocarcinoma. Although the liver is the major site of injury, AFB1-induced tumors have been experimentally produced in the lungs, kidneys, and colons of rodents.

Aflatoxin B2

Aflatoxin B2 is a naturally occurring mycotoxin produced by species of the mold Aspergillus, which can be found in legumes, corn, soybeans, rice, milk, and cheese. The highest levels of aflatoxin contamination are always associated with post-harvest spoilage, when commodities are stored with an inappropriate moisture content and temperature.

However, the aflatoxin contamination is not simply a problem of poor storage but can occur in the field before the crop is harvested. The spores of these species of Aspergillus can land on the stigma of the developing plant, and then germinate and penetrate to the immature seed tissue just as if they were pollen grains. The mold can establish an endophytic growth within the tissues of the plant without causing any perceptible harm to the plant.

Aflatoxin G1

Aflatoxin G1 (AFG1) is one of the four major known aflatoxins naturally produced by the Aspergillus species. Aflatoxins may be present in a wide range of food commodities, particularly cereals, oil seeds, spices and tree nuts like maize, groundnuts (peanuts), pistachios, chilis, black pepper, dried fruit and figs. It has also been detected in milk and milk products. Less is known about the chronic toxicity of aflatoxin G1, but these are also thought to be carcinogens, though probably a little less potent than B1.[13]

Aflatoxin G2

Aflatoxin G2 (AFG2) is one of the four major known aflatoxins naturally produced by the Aspergillus species. It is considered less toxic than AFB1, AFB2, and AFG1. In vivo animal studies have shown that AFG2 targets the liver and kidneys in chicken and sheep.[13]

Trichothecenes

Trichothecenes are a very large family of chemically related mycotoxins produced by various species of Fusarium, Myrothecium, Trichoderma, Trichothecium, Cephalosporium, Verticimonosporium, and Stachybotrys.

These mycotoxins are commonly found on food, such as wheat, oats, or maize, but can be produced by molds and fungi which can grow indoors on water-damaged materials. They can also be absorbed through dermal contact.

Trichothecenes accelerate the production of reactive oxygen species in cells and inhibit protein synthesis in a number of ways. This leads to cell damage or cell death.

Roridin E *(Fusarium, Myrothecium, Stachybotrys)*

Roridin E is a well-known macrocyclic trichothecene mycotoxin produced by various species of Fusarium, Myrothecium, Trichoderma, Trichothecium, Cephalosporium, Verticimonosporium, and Stachybotrys. They are produced on many different grains like wheat, oats, or maize by various Fusarium species. Some molds that produce trichothecene mycotoxins, such as Stachybotrys chartarum, can grow in damp indoor environments and may contribute to health problems among building occupants.[6]

Verrucarin A *(Stachybotrys, Fusarium, Myrothecium)*

Verrucarin A is macrocyclic trichothecene produced largely by Myrothecium, Stachybotrys and Fusarium. This toxin has a wide range of antiviral, antifungal, and antibacterial activity. Trichothecenes are generally produced on many different grains like wheat, oats or maize.

Deoxynivalenol (Vomitoxin/DON) *(Fusarium)*

Deoxynivalenol (DON), also known as Deoxynivalenol, a tricothecene mycotoxin, is produced by several species of Fusarium. DON has been associated with outbreaks of acute gastrointestinal illness in humans. The FDA advisory level for DON for human consumption is 1 ppm.

Nivalenol (NIV) *(Fusarium)*

Produced by the mold genus Fusarium, the type B trichothecenes, nivalenol (NIV) and their acetylated precursors are often found contaminating cereal staples, posing a potential threat to public health that is still incompletely understood. Trichothecenes are very resistant to milling and processing, so they can enter human food products easily. NIV is not found in food as commonly as DON; however, it demonstrates higher toxicity in animal studies. The toxicity of NIV is often compared to the toxicity of DON; however, the amount of toxicological data on NIV impact is much lower compared to DON.[19]

Diacetoxyscirpenol (DAS) *(Fusarium)*

Diacetoxyscirpenol (DAS), also known as anguidine, is a type A trichothecene mycotoxin primarily produced by Fusarium fungi. Trichothecenes are known as major contaminants of cereals and cereal-containing foods. DAS has been detected in agricultural products worldwide and persists in products after processing. In humans as well as in animals, DAS consumption has been shown to induce haematological disorders (neutropenia, aplastic anemia). In the published literature, DAS has mainly been reported in various cereal grains (principally wheat, sorghum, maize, barley, and oats) and cereal products, but also in potato products, soybeans, and coffee. The highest levels have been reported for wheat, sorghum, and coffee. DAS has been found to co-occur with many other mycotoxins in grains and grain-based products, in particular Fusarium toxins including type A and B trichothecenes, and zearalenone.[20]

T-2 toxin *(Fusarium)*

T-2 Toxin is a tricothecene produced by species of Fusarium and is one of the rare and deadlier toxins. If ingested in sufficient quantity, T-2 toxin can severely damage the entire digestive tract and cause rapid death due to internal hemorrhage. T-2 has been implicated in the human diseases alimentary toxic aleukia and pulmonary hemosiderosis. Damage caused by T-2 toxin is often permanent.

Satratoxin G *(Stachybotrys chartarum)*

Satratoxin G is a macrocyclic trichothecene mycotoxin produced by commonly called black mold, or Stachybotrys chartarum, that contributes to disorders associated with water-damaged buildings. It is a potent inhibitor of protein translation that initiates both inflammatory gene expression and apoptosis in vitro after upstream activation of mitogen-activated protein kinases (MAPKs). These water-soluble mycotoxins could produce airborne particles which could facilitate entry and release into respiratory airway tissue that may selectively induce apoptosis in olfactory sensory neurons in the nose (rhinitis) and brain (mild focal encephalitis).[21]

Satratoxin H *(Stachybotrys chartarum)*

Satratoxin H is a trichothecene mycotoxin that has been recognized as one of the potential etiologic agents in outbreaks of sick building syndromes. Satratoxin H potently inhibits protein synthesis and thymocyte proliferation and also can cause diseases such as an immune dysfunction and idiopathic pulmonary hemorrhage in infants. Recent studies have shown a possible relationship between trichothecenes and disorders of the central nervous system, including severe neuronal death.[22]

Isosatratoxin F *(Stachybotrys chartarum)*

Isosatratoxin F is another trichothecene produced by Stachybotrys chartarum. Several animal studies have shown that isosatratoxin F can cause nasal and pulmonary toxicity when administered intranasally or intratracheally. They showed that pulmonary alveolus cells were injured following intratracheal instillation of isosatratoxin F, with marked changes in surfactant synthesis and secretion.[23]

Roridin A *(Stachybotrys chartarum)*

Roridin A mycotoxin is one of the important macrocyclic trichothecenes, produced on foodstuffs such as corn, rice, wheat, and other crops. Trichothecence mycotoxins prevent polypeptide chain initiation or elongation and interact with the enzyme peptidyl transferase. Both humans and animals suffer from several pathologies due to intoxication after consumption of foodstuffs contaminated with trichothecences and the conditions have been named differently according to the causative fungus.[24]

Roridin H *(Stachybotrys chartarum)*

Roridin H is produced mainly by Stachybotrys and categorized as a trichothecene mycotoxin. There are reports showing the involvement of these trichothecenes in the development of 'sick building syndrome.' These trichothecenes were found in air samples in the ventilation systems of private houses and office buildings, and on the walls of houses with high humidity. The symptoms of airborne toxicosis disappeared when the buildings and ventilation systems were thoroughly cleaned.[25]

Roridin L-2

Roridin L2 is the putative biosynthetic precursor of Satratoxin G. It is a common trichothecene produced by S. chartarum isolates from water-damaged homes. Due to structural differences, roridin L2 possesses little in vitro or in vivo toxic activity as compared to SG.[26]

Verrucarin J *(Stachybotrys chartarum)*

Verrucarin J is a trichothecene produced by Stachybotrys chartarum. They can grow in damp indoor environments and may contribute to health problems among building occupants. These Trichothecenes are lipophilic and thus the route of exposure can easily be through the skin, gut, and pulmonary mucosa.[27]

Ochratoxin A *(Aspergillus, Penicillium)*

Members of the ochratoxin A family have been found as metabolites of many different species of Aspergillus and Penicillium. The level of Ochratoxin A production was also influenced by the substrate on which the molds grow as well as the moisture level, temperature, and presence of competitive microflora interactions to influence the level of toxin produced.
Ochratoxin A has been found in barley, oats, rye, wheat, coffee beans, and other plant products, with barley having a particularly high likelihood of contamination. Ochratoxin has been detected in blood and other animal tissues and in milk, including human milk.
Ochratoxin A is a nephrotoxin to all animal species studied to date and is most likely toxic to humans, who have the longest half-life for its elimination of any of the species. It is frequently found in pork intended for human consumption. Ochratoxin is believed to be responsible for a porcine nephropathy that has been studied intensively in the Scandinavian countries. The disease is endemic in Denmark, where rates of porcine nephropathy and ochratoxin contamination in pig feed are highly correlated. In addition to being a nephrotoxin, animal studies indicate that ochratoxin A is a liver toxin, an immune suppressant, a potent teratogen, and a carcinogen.[3]

Sterigmatocystin *(Aspergillus, Penicillium, Bipolaris)*

Sterigmatocystin, a related dihydrofuran toxin, is a late metabolite in the aflatoxin pathway and is also produced as a final biosynthetic product by a number of species such as Aspergillus, Penicillium, and Bipolaris. STC is a possible human carcinogen (2B) according to IARC classification and showed immunotoxic and immunomodulatory activity, together with mutagenic effects.
It might be found in numerous substrates, from foods and feeds to chronically damp building materials and indoor dust. Due to the structural similarities, aflatoxins and STC share relevant toxic effects, including genotoxicity and carcinogenicity. However, in contrast to aflatoxins, only limited information on occurrence and toxicity of STC is available. Liver and kidneys are the target organs of acute toxicity of STC. However, the acute oral toxicity is relatively low (range between 120 and 166 mg/kg body weight).[4]

Satratoxin H *(Stachybotrys chartarum)*

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Zearalenone *(Fusarium)*

Zearalenone (ZEA) is a non-steroidal estrogenic mycotoxin. It is produced principally by Fusarium molds, and consequently occurs wherever DON occurs, most notably as a contaminant of maize, wheat, barley, oats, rye, sorghum, millet, and rice. ZEA and its metabolites can bind to estrogen receptors, resulting in various changes in the reproductive organs. In addition, however, ZEA is a competitive substrate for enzymes involved in steroid synthesis and metabolism and therefore has the potential to act as an endocrine disruptor.[5]

Enniatin B1 *(Fusarium)*

Mycotoxin enniatin B (ENN B) is a secondary metabolism product by Fusarium fungi. It is a well-known antibacterial, antihelmintic, antifungal, herbicidal, and insecticidal compound. It has been found as a contaminant in cereal grains, animal feeds, and several food commodities worldwide, co-occurring with other mycotoxins. Moreover, they are commonly found in fish, dried fruits, nuts, spices, cocoa, and coffee products.
Food processing techniques such as cooking, baking, frying, roasting, etc. do not affect their chemical structure; so, detoxification strategies to mitigate the risks of ENNs presence in foods and feed may be difficult. Several in vitro and in vivo studies have revealed that ENN B toxicity involves the inhibition of acyl-CoA: cholesterol acyl transferase (ACAT) activity and oxidative stress. ENN B also exerts cytotoxic activities by inducing mitochondrial modifications and cell cycle disruption, finally resulting in apoptotic cell death. Moreover, it produces adrenal endocrine toxicity. A recent study reports a potential anticancer activity. Nevertheless, regulatory limits have not yet been defined, due to a lack of complete toxicity data.[8]

Fumonisins B1 *(Fusarium)*

Fusarium is one of the most prevalent fungi associated with contamination of corn and other agricultural products throughout the world. Many different fumonisins have so far been reported, and they have been grouped into four main categories (A, B, C, and P). The most abundant of the fumonisins is fumonisin B1 (FB1). They can also be found in moisture-damaged buildings, and, therefore, exposure of humans to Fusarium mycotoxins including FB1 may take place. FB1 bears a clear structural similarity to the cellular sphingolipids, and this similarity has been shown to disturb the metabolism of sphingolipids by inhibiting a key enzyme in sphingolipid biosynthesis. FB1 is neurotoxic, hepatotoxic, and nephrotoxic in animals, and it has been classified as a possible carcinogen to humans. The cellular mechanisms behind FB1-induced toxicity include the induction of oxidative stress, apoptosis, and cytotoxicity, as well as alterations in cytokine expression.[9]

Fumonisins B2 *(Fusarium)*

Fumonisin B2 is a mycotoxin produced by Fusarium growing on moldy corn (maize) grain. FB2 and Fumonisin B3 (FB3) occur in lower concentrations than FB1. FB1 and FB2 are approximately equal in structure and toxicity but naturally occur in a ratio of about 3:1 for FB1/FB2, thus has less toxicity than FB1.[9]

Fumonisins B3 *(Fusarium)*

Fumonisin B3 is a mycotoxin produced mainly by Fusarium, that belongs to the Fumonisins family, one of the most prevalent fungi of maize-based crops. Fumonisin B3 is the third most abundant fumonisin found in contaminated maize. However, FB3 is less toxic than FB1 and FB2.[9]

Citrinin *(Penicillium)*

Citrinin (CTN) is a nephrotoxic mycotoxin mainly produced by Penicillium, although other genera such as Aspergillus and Monascus are also known to produce these toxins. CTN occurs in different plant products, especially in grains, and also in beans, fruit, vegetables, herbs, and spices.
Often, the co-occurrence with other mycotoxins is observed, especially ochratoxin A (OTA). It is a known fact that CTN occurs during fermentation of red mould rice (also referred to as red yeast rice) as a secondary metabolite of Monascus purpureus. Red mould rice has been used for lowering lipoprotein levels in blood and also as a food dye for centuries. Besides its nephrotoxicity, which has been proved by various studies, there is also proof that CTN is involved in induction of apoptosis through oxidative stress, although the precise regulatory mechanism is yet to be determined.[10]

Patulin *(Penicillium)*

Patulin is a toxic chemical contaminant produced by several species of mold, especially within Aspergillus, Penicillium and Byssochlamys. Penicillium expansum, the blue mold that causes soft rot of apples, pears, cherries, and other fruits, is recognized as one of the most common offenders in patulin contamination.[3] It is the most common mycotoxin found in apples and apple-derived products such as juice, cider, compotes, and other food intended for young children. Exposure to this mycotoxin is associated with immunological, neurological and gastrointestinal outcomes.[11]
Several studies have revealed its mutagenicity, teratogenicity, chromosomal aberration, DNA strand damage, and micronuclei formation in mammalian cells. However, pieces of evidence for the carcinogenic potential of PAT in the animal model are not sufficient. Based on the available data, the presence of PAT can be used as a quality control parameter, as its detection in apple-derived food such as juices, ciders, and concentrates indicated that moldy apples were used in the production of juices and the accumulation of PAT within the body may pose toxicological threats. For this reason, the problem of detecting even low levels of PAT in apple juices continues to receive attention. Because apple juice is such a popular beverage and the possibility for life-long exposure exists, PAT will likely remain important to apple processors and governments interested in monitoring the quality of apple juices and products.[12]

Gliotoxin *(Aspergillus)*

Gliotoxin is mainly produced by Aspergillus and sometimes by Penicillium species. Gliotoxin can be produced by common indoor molds and enter the human body via inhalation of mycotoxin containing spores and particulates.[14] Gliotoxin produced by Aspergillus fumigatus could promote immunosuppression by inhibiting/interfering with the activation of transcription factors that are involved in T cell activation. In addition to targeting immune system, gliotoxins have adverse effects on the kidneys and liver.[15]

Mycophenolic Acid *(Penicillium)*

Mycophenolic Acid (MPA) is a mycotoxin produced by a number of Penicillium species. Penicillium brevicompactum, a species able to produce mycophenolic acid (MPA), has also been frequently identified in indoor environments that can be found on building materials, in dust, and in air samples.[16] It usually suppresses the immune system by inhibiting the proliferation of B and T lymphocytes.

Dihydrocitrinone (Aspergillus, Penicillium, Monascus)

Dihydrocitrinone (DH-CIT) is the main metabolite of Citrinin (CTN), a mycotoxin produced mainly by Penicillium and also by Aspergillus. In vitro studies have shown that DH-CIT is clearly less cytotoxic compared to parental CTN.[17] CTN mainly acts as a nephrotoxic compound.

Chaetoglobosin A *(Chaetomium globosum)*

Chaetomium globosum is a fungus frequently found in water-damaged buildings and produces a mycotoxin called Chaetoglobosin A. Relatively low levels of this compound have been shown to be lethal to various tissue culture cell lines. The presence of Chaetoglobosin A can be lethal to mammalian cells which act by binding to actin, leading to inhibition of cell division, locomotion, and formation of cell surface projections.[18]

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