

Mycotoxin List

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Mold Help Organization

Some Common Mycotoxins

Mycotoxin Descriptions

Mycotoxins produced by indoor fungi

Molds can produce other secondary metabolites (see list in table below) such as antibiotics and mycotoxins. Antibiotics are isolated from mold (and some bacterial) cultures and some of their bacteriotoxic or bacteriostatic properties are exploited medicinally to combat infections.

Mycotoxins are also products of secondary metabolism of molds. They are not essential to maintaining the life of the mold cell in a primary way (at least in a friendly world), such as obtaining energy or synthesizing structural components, informational molecules or enzymes. They are products whose function seems to be to give molds a competitive advantage over other mold species and bacteria. 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. Of course they are also toxic to the cells of higher plants and animals, including humans.

Mycotoxins vary in specificity and potency for their target cells, cell structures or cell processes by species and strain of the mold that produces them. Higher organisms are not specifically targeted by mycotoxins, but seem to be caught in the crossfire of the biochemical warfare among mold species and molds and bacteria vying for the same ecological niche.

Not all molds produce mycotoxins, but numerous species do (including some found indoors in contaminated buildings). Toxigenic molds vary in their mycotoxin production depending on the substrate on which they grow (Jarvis, 1990). The spores, with which the toxins are primarily associated, are cast off in blooms that vary with the mold's diurnal, seasonal and life cycle stage (Burge, 1990; Yang, 1995). The presence of competitive organisms may play a role, as some molds grown in monoculture in the laboratory lose their toxic potency (Jarvis, 1995). Until relatively recently, mold poisons were regarded with concern primarily as contaminants in foods.

More recently concern has arisen over exposure to multiple mycotoxins from a mixture of mold spores growing in wet indoor environments. Health effects from exposures to such mixtures can differ from those related to single mycotoxins in controlled laboratory exposures. Indoor exposures to toxigenic molds resemble field exposures of animals more closely than they do controlled experimental laboratory exposures. Animals in controlled laboratory exposures are healthy, of the same age, raised under optimum conditions, and have only the challenge of known doses of a single toxic agent via a single exposure route. In contrast, animals in field exposures are of mixed ages, and

states of health, may be living in less than optimum environmental and nutritional conditions, and are exposed to a mixture of toxic agents by multiple exposure routes. Exposures to individual toxins may be much lower than those required to elicit an adverse reaction in a small controlled exposure group of ten animals per dose group. The effects from exposure may therefore not fit neatly into the description given for any single toxin, or the effects from a particular species, of mold. Field exposures of animals to molds (in contrast to controlled laboratory exposures) show effects on the immune system as the lowest observed adverse effect. Such immune effects are manifested in animals as increased susceptibility to infectious diseases (Jakab et al., 1994). It is important to note that almost all mycotoxins have an immunosuppressive effect, although the exact target within the immune system may differ. Many are also cytotoxic, so that they have route of entry effects that may be damaging to the gut, the skin or the lung. Such cytotoxicity may affect the physical defense mechanisms of the respiratory tract, decreasing the ability of the airways to clear particulate contaminants (including bacteria or viruses), or damage alveolar macrophages, thus preventing clearance of contaminants from the deeper lung. The combined result of these activities is to increase the susceptibility of the exposed person to infectious disease, and to reduce his defense against other contaminants. They may also increase susceptibility to cancer.

Because indoor samples are usually comprised of a mixture of molds and their spores, it has been suggested that a general test for cytotoxicity be applied to a total indoor sample to assess the potential for hazard as a rough assessment (Gareis, 1995).

The following summary of toxins and their targets is adapted from Smith and Moss (1985), with a few additions from the more recent literature. While this compilation of effects does not describe the effects from multiple exposures, which could include synergistic effects, it does give a better idea of possible results of mycotoxin exposure to multiple molds indoors.

- Vascular system (increased vascular fragility, hemorrhage into body tissues, or from lung, e. g., aflatoxin, satratoxin, roridins).
- Digestive system (diarrhea, vomiting, intestinal hemorrhage, liver effects, i. e., necrosis, fibrosis: aflatoxin; caustic effects on mucous membranes: T-2 toxin; anorexia: vomitoxin).
- Respiratory system: respiratory distress, bleeding from lungs e. g., trichothecenes.
- Nervous system, tremors, incoordination, depression, headache, e. g., tremorgens, trichothecenes.
- Cutaneous system: rash, burning sensation sloughing of skin, photosensitization, e. g., trichothecenes.
- Urinary system, nephrotoxicity, e. g. ochratoxin, citrinin.
- Reproductive system; infertility, changes in reproductive cycles, e. g. T-2 toxin, zearalenone.
- Immune system: changes or suppression: many mycotoxins.

It should be noted that not all mold genera have been tested for toxins, nor have all species within a genus necessarily been tested. Conditions for toxin production vary with cell and diurnal and seasonal cycles and substrate on which the mold grows, and those conditions created for laboratory culture may differ from those the mold encounters in its environment. Toxicity can arise from exposure to mycotoxins via inhalation of mycotoxin-containing mold spores or through skin contact with the toxigenic molds (Forgacs, 1972;

Croft et al., 1986; Kemppainen et al., 1988 -1989). A number of toxigenic molds have been found during indoor air quality investigations in different parts of the world. Among the genera most frequently found in numbers exceeding levels that they reach outdoors are *Aspergillus*, *Penicillium*, *Stachybotrys*, and *Cladosporium* (Burge, 1986; Smith et al., 1992; Hirsh and Sosman, 1976; Verhoeff et al., 1992; Miller et al., 1988; Gravesen et al., 1999).

Some Common Mycotoxins and the Organisms that Produce them

Mycotoxin	Organism
Acetoxyscirpenediol	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Acetyldeoxynivalenol	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Acetylneosolaniol	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Acetyl T-2 toxin	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Aflatoxin	<i>Aspergillus flavus</i> , <i>A. parasiticus</i>
Aflatrem	<i>Aspergillus flavus</i>
Altenuic acid	<i>Alternaria alternata</i>
Alternariol	<i>Alternaria alternata</i>
Austdiol	<i>Aspergillus ustus</i>
Austamide	<i>Aspergillus ustus</i>
Austocystin	<i>Aspergillus ustus</i>
Avenacein +1	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Beauvericin +2	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Bentenolide	<i>Monographella nivalis</i>
Brevianamide	<i>Aspergillus ustus</i>
Butenolide	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Calonectrin	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Chaetoglobosin	<i>Chaetomium globosum</i>
Citrinin	<i>Aspergillus carneus</i> , <i>A. terreus</i> , <i>Penicillium citrinum</i> , <i>P. hirsutum</i> , <i>P. verrucosum</i>
Citreoviridin	<i>Aspergillus terreus</i> , <i>Penicillium citreoviride</i>
Cochliodinol	<i>Chaetomium cochliodes</i>
Crotocin	<i>Acremonium crotocinigenum</i>
Cytochalasin E	<i>Aspergillus clavatus</i>
Cyclopiazonic acid	<i>Aspergillus versicolor</i>

Deacetylcalonecetrin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Deoxynivalenol diacetate	<i>Fusarium moniliforme, and F. nivale</i>
Deoxynivalenol monoacetate	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Diacetoxyscirpenol	<i>Fusarium moniliforme, F. equiseti</i>
Destruxin B	<i>Aspergillus ochraceus</i>
Enniatins	<i>Fusarium moniliforme, F. avenaceum, F. roseum, F. solani, and F. nivale</i>
Fructigenin +1	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. roseum</i>
Fumagilin	<i>Aspergillus fumigatus</i>
Fumonisin B1	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. nivale</i>
Fusaric acid	<i>Fusarium moniliforme</i>
Fusarin	<i>Fusarium moniliforme</i>
Gliotoxin	<i>Alternaria, Aspergillus fumigatus, Penicillium</i>
HT-2 toxin	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. nivale</i>
Ipomeanine	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. nivale</i>
Islanditoxin	<i>Penicillium islandicum</i>
Lateritin +1	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. nivale</i>
Lycomarasmin +1	<i>Fusarium moniliforme</i>
Malformin	<i>Aspergillus niger</i>
Maltoryzine	<i>Aspergillus spp.</i>
Moniliformin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Monoacetoxyscirpenol	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Neosolaniol	<i>Fusarium moniliforme, F. solani, F. culmorum, F. avenaceum, and F. roseum</i>
Nivalenol	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
NT-1 toxin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
NT-2 toxin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. , F. solani, avenaceum, F. roseum, and F. nivale</i>
Ochratoxin	<i>Aspergillus ochraceus, Penicillium viridictum</i>

Oxalic acid	<i>Aspergillus niger</i>
Patulin	<i>Aspergillus clavatus</i> , <i>Penicillium expansum</i> , <i>Botrytis</i> , <i>P. roquefortii</i> , <i>P. claviforme</i> , <i>P. griseofulvum</i>
Penicillic acid	<i>Aspergillus ochraceus</i>
Penitrem	<i>Penicillium crustosum</i>
Roridin E	<i>Myrothecium roridum</i> , <i>M. verrucaria</i> , <i>Dendrodochium spp.</i> , <i>Cylindrocarpon spp.</i> , <i>Stachybotrys spp.</i>
Rubratoxin	<i>Penicillium rubrum</i>
Rubroskyrin	<i>Penicillium spp.</i>
Rubrosulphin	<i>Penicillium viridicatum</i>
Rugulosin	<i>Penicillium brunneum</i> , <i>P. kloeckeri</i> , <i>P. rugulosum</i>
Sambucynin +1	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. solani</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Satratoxins, F,G,H	<i>Stachybotrys chartarum</i> , <i>Trichoderma viridi</i>
Scirpentriol	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. solani</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Slaframine	<i>Rhizoctonia leguminicola</i>
Sterigmatocystin	<i>Aspergillus flavus</i> , <i>A. nidulans</i> , <i>A. versicolor</i> , <i>Penicillium rugulosum</i>
T-1 toxin	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. culmorum</i> , <i>F. solani</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
T-2 toxin	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. culmorum</i> , <i>F. solani</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Triacetoxyscirpendiol	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Trichodermin	<i>Trichoderma viride</i>
Trichothecin	<i>Trichothecium roseum</i>
Trichoverrins	<i>Stachybotrys chartarum</i>
Trichoverrols	<i>Stachybotrys chartarum</i>
Tryptoquivalene	<i>Aspergillus clavatus</i>
Verrucarin	<i>Myrothecium verrucaria</i> , <i>Dendrodochium spp.</i> , <i>Stachybotrys chartarum</i>
Verruculogen	<i>Aspergillus fumigatus</i> , <i>Stachybotrys chartarum</i>
Viopurpurin	<i>Trichophyton spp.</i> , <i>Penicillium viridicatum</i>
Viomellein	<i>Aspergillus spp.</i> , <i>Penicillium aurantiogriseum</i> , <i>P. crustosum</i> , <i>P. viridicatum</i>
Viriditoxin	<i>Aspergillus fumigatus</i>
Xanthocillin	<i>Eurotium chevalieri</i>
Yvanicin+1	<i>Fusarium culmorum</i> , <i>F. graminearum</i> , <i>F. oxysporum</i> , <i>F. roseum</i> , <i>F. moniliforme</i> , <i>F. avenaceum</i> , <i>F. equiseti</i> , and

	<i>F. nivale</i>
Zearalenone	<i>Fusarium culmorum, F. graminearum, F. oxysporum, F. roseum, F. moniliforme, F. avenaceum, F. equiseti, and F. nivale</i>
Mycotoxin	<i>Organism</i>
Acetoxyscirpenediol	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Acetyldeoxynivalenol	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Acetylneosolaniol	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Acetyl T-2 toxin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Aflatoxin	<i>Aspergillus flavus, A. parasiticus</i>
Aflatrem	<i>Aspergillus flavus</i>
Altenuic acid	<i>Alternaria alternata</i>
Alternariol	<i>Alternaria alternata</i>
Austdiol	<i>Aspergillus ustus</i>
Austamide	<i>Aspergillus ustus</i>
Austocystin	<i>Aspergillus ustus</i>
Avenacein +1	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Beauvericin +2	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Bentenolide	<i>Monographella nivalis</i>
Brevianamide	<i>Aspergillus ustus</i>
Butenolide	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Calonectrin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Chaetoglobosin	<i>Chaetomium globosum</i>
Citrinin	<i>Aspergillus carneus, A. terreus, Penicillium citrinum, P. hirsutum, P. verrucosum</i>
Citreoviridin	<i>Aspergillus terreus, Penicillium citreoviride</i>
Cochliodinol	<i>Chaetomium cochliodes</i>
Crotocin	<i>Acremonium crotocinigenum</i>
Cytochalasin E	<i>Aspergillus clavatus</i>
Cyclopiazonic acid	<i>Aspergillus versicolor</i>
Deacetylcalonectrin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Deoxynivalenol diacetate	<i>Fusarium moniliforme, and F. nivale</i>

Deoxynivalenol monoacetate	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Diacetoxyscirpenol	<i>Fusarium moniliforme, F. equiseti</i>
Destruxin B	<i>Aspergillus ochraceus</i>
Enniatins	<i>Fusarium moniliforme, F. avenaceum, F. roseum, F. solani, and F. nivale</i>
Fructigenin +1	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. roseum</i>
Fumagilin	<i>Aspergillus fumigatus</i>
Fumonisin B1	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. nivale</i>
Fusaric acid	<i>Fusarium moniliforme</i>
Fusarin	<i>Fusarium moniliforme</i>
Gliotoxin	<i>Alternaria, Aspergillus fumigatus, Penicillium</i>
HT-2 toxin	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. nivale</i>
Ipomeanine	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. nivale</i>
Islanditoxin	<i>Penicillium islandicum</i>
Lateritin +1	<i>Fusarium moniliforme, F. culmorum, F. avenaceum, and F. nivale</i>
Lycomarasmin +1	<i>Fusarium moniliforme</i>
Malformin	<i>Aspergillus niger</i>
Maltoryzine	<i>Aspergillus spp.</i>
Moniliformin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Monoacetoxyscirpenol	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
Neosolaniol	<i>Fusarium moniliforme, F. solani, F. culmorum, F. avenaceum, and F. roseum</i>
Nivalenol	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
NT-1 toxin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. avenaceum, F. roseum, and F. nivale</i>
NT-2 toxin	<i>Fusarium moniliforme, F. equiseti, F. oxysporum, F. culmorum, F. , F. solani, avenaceum, F. roseum, and F. nivale</i>
Ochratoxin	<i>Aspergillus ochraceus, Penicillium viridictum</i>
Oxalic acid	<i>Aspergillus niger</i>
Patulin	<i>Aspergillus clavatus, Penicillium expansum, Botrytis, P. roquefortii, P. claviforme, P. griseofulvum</i>
Penicillic acid	<i>Aspergillus ochraceus</i>

Penitrem	<i>Penicillium crustosum</i>
Roridin E	<i>Myrothecium roridum</i> , <i>M. verrucaria</i> , <i>Dendrodochium spp.</i> , <i>Cylindrocarpon spp.</i> , <i>Stachybotrys spp.</i>
Rubratoxin	<i>Penicillium rubrum</i>
Rubroskyrin	<i>Penicillium spp.</i>
Rubrosulphin	<i>Penicillium viridicatum</i>
Rugulosin	<i>Penicillium brunneum</i> , <i>P. kloeckeri</i> , <i>P. rugulosum</i>
Sambucynin +1	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. solani</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Satratoxins, F,G,H	<i>Stachybotrys chartarum</i> , <i>Trichoderma viridi</i>
Scirpentriol	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. oxysporum</i> , <i>F. culmorum</i> , <i>F. solani</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Slaframine	<i>Rhizoctonia leguminicola</i>
Sterigmatocystin	<i>Aspergillus flavus</i> , <i>A. nidulans</i> , <i>A. versicolor</i> , <i>Penicillium rugulosum</i>
T-1 toxin	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. culmorum</i> , <i>F. solani</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
T-2 toxin	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. culmorum</i> , <i>F. solani</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Triacetoxyscirpendiol	<i>Fusarium moniliforme</i> , <i>F. equiseti</i> , <i>F. avenaceum</i> , <i>F. roseum</i> , and <i>F. nivale</i>
Trichodermin	<i>Trichoderma viride</i>
Trichothecin	<i>Trichothecium roseum</i>
Trichoverrins	<i>Stachybotrys chartarum</i>
Trichoverrols	<i>Stachybotrys chartarum</i>
Tryptoquivalene	<i>Aspergillus clavatus</i>
Verrucaric acid	<i>Myrothecium verrucaria</i> , <i>Dendrodochium spp.</i> , <i>Stachybotrys chartarum</i>
Verruculogen	<i>Aspergillus fumigatus</i> , <i>Stachybotrys chartarum</i>
Viopurpurin	<i>Trichophyton spp.</i> , <i>Penicillium viridicatum</i>
Viomellein	<i>Aspergillus spp.</i> , <i>Penicillium aurantiogriseum</i> , <i>P. crustosum</i> , <i>P. viridicatum</i>
Viriditoxin	<i>Aspergillus fumigatus</i>
Xanthocillin	<i>Eurotium chevalieri</i>
Yavanicin+1	<i>Fusarium culmorum</i> , <i>F. graminearum</i> , <i>F. oxysporum</i> , <i>F. roseum</i> , <i>F. moniliforme</i> , <i>F. avenaceum</i> , <i>F. equiseti</i> , and <i>F. nivale</i>
Zearalenone	<i>Fusarium culmorum</i> , <i>F. graminearum</i> , <i>F. oxysporum</i> , <i>F. roseum</i> , <i>F. moniliforme</i> , <i>F. avenaceum</i> , <i>F. equiseti</i> , and <i>F. nivale</i>

Fungi are ubiquitous to the environment and primarily saprophytic, using nonliving organic material as a nutrient source for growth and reproduction. Many of these saprophytes can colonize organic water-damaged building materials. During the digestion process fungi secrete enzymes into the nutrient source to break down complex compounds into simpler compounds, which are taken up by the fungi and digested. The digested nutrients are classified into two categories, primary and secondary metabolites. The primary metabolites consist of cellulose and other compounds that are used for energy to grow and reproduce.

The secondary metabolites, called mycotoxins, are produced to give fungi a competitive edge against other microorganisms, including other fungi. There are over 200 recognized mycotoxins, however, the study of mycotoxins and their health effects on humans is in its infancy and many more are waiting to be discovered. Many mycotoxins are harmful to humans and animals when inhaled, ingested or brought into contact with human skin. Mycotoxins can cause a variety of short term as well as long-term health effects, ranging from immediate toxic response to potential long-term carcinogenic and teratogenic effects. Symptoms due to exposure to mycotoxins include dermatitis, cold and flu symptoms, sore throat, headache, fatigue, diarrhea, and impaired or altered immune function, which may lead to opportunistic infection. Historically, mycotoxins have been a persistent problem to farmers and the animal husbandry industry in Eastern Europe and developing countries. Mycotoxins are a known agent in biological warfare as a moderate illness compared to the other biologicals.

Recently, however, research has implicated many toxin-producing fungi, such as *Stachybotrys*, *Penicillium*, *Aspergillus* and *Fusarium* species, to indoor air quality problems and building related illnesses. Inhalation of mycotoxin producing fungi in contaminated buildings is the most significant exposure, however, dermal contact from handling contaminated materials and the chance of ingesting toxin containing spores through eating, drinking and smoking is likely to increase exposure in a contaminated environment. Recent advances in technology have given laboratories the ability to test for specific mycotoxins without employing cost-prohibitive gas chromatography or high performance liquid chromatography techniques. Currently, surface, bulk, food and feeds, and air samples can be analyzed relatively inexpensively for the following mycotoxins:

Aflatoxin

Aflatoxin is one of the most potent carcinogens known to man and has been linked to a wide variety of human health problems. The FDA has established maximum allowable levels of total aflatoxin in food commodities at 20 parts per billion. The maximum level for milk products is even lower at 0.5 parts per billion. Primarily *Aspergillus* species fungi produce aflatoxin.

Ochratoxin

Ochratoxin is primarily produced by species of *Penicillium* and *Aspergillus*. Ochratoxin is damaging to the kidneys and liver and is also a suspected carcinogen. There is also evidence that it impairs the immune system.

T-2 Toxin

T-2 Toxin is a tricothecene produced by species of *Fusarium* and is one of the more deadly 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.

Fumonisin

Fumonisin is a toxin associated with species of *Fusarium*. Fumonisin is commonly found in corn and corn-based products, with recent outbreaks of veterinary mycotoxicosis occurring in Arizona, Indiana, Kentucky, North Carolina, South Carolina, Texas and Virginia. The animals most affected were horses and swine, resulting in dozens of deaths. Fumonisin toxin causes "crazy horse disease", or leukoencephalomalacia, a liquefaction of the brain. Symptoms include blindness, head butting and pressing, constant circling and ataxia, followed by death. Chronic low-level exposure in humans has been linked to esophageal cancer. The American Association of Veterinary Laboratory Diagnosticians (AAVLD) advisory levels for fumonisin in horse feed is 5 ppm.

Vomitoxin or Deoxynivalenol(DON)

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

Zearalenone

Zearalenone is also a mycotoxin produced by *Fusarium* molds. Zearalenone toxin is similar in chemical structure to the female sex hormone estrogen and targets the reproductive organs.

Other mycotoxins of clinical significance are as follows:

Citrinin

Citrinin is a nephrotoxin produced by *Penicillium* and *Aspergillus* species. Renal damage, vasodilatation, and bronchial constriction are some of the health effects associated with this toxin.

Alternariol

Alternariol cytotoxic compound derived from *Alternaria alternata*

Satratoxin H

Satratoxin H is a macrocyclic tricothecene produced by *Stachybotrys chartarum*, *Trichoderma viridi* and other fungi. High doses or chronic low doses are lethal. This toxin is abortogenic in animals and is believed to alter immune system function and makes affected individuals more susceptible to opportunistic infection.

Gliotoxin

Gliotoxin is an immunosuppressive toxin produced by species of *Alternaria*, *Penicillium* and *Aspergillus*.

Patulin

Patulin is a mycotoxin produced by *Penicillium*, *Aspergillus* and a number of other genera of fungi. It is believed to cause hemorrhaging in the brain and lungs and is usually associated with apple and grape spoilage.

Sterigmatocystin

Sterigmatocystin is a nephrotoxin and a hepatotoxin produced by *Aspergillus versicolor*. This toxin is also considered to be carcinogenic. Other mycotoxins include - Penicillic acid, roquefortine, cyclopiazonic acid, verrucosidin, rubratoxins A and B, PR toxin, luteoskyrin, cyclochlorotine, rugulosin, erythrokyrine, secalonic acid D, viridicatumtoxin, kojic acid, xanthomegnin, viomellein, chaetoglobosin C, echinulin, flavoglucin, versicolorin A, austamide, maltoyzine, aspergillic acid, paspaline, aflatrem, fumagillin nigragillin chlamydosporol, isotrichodermin and many more.

As discussed there are many mycotoxins that can cause adverse health effects and even death in humans. The synergistic effect of exposure to multiple mycotoxins simultaneously is very poorly understood. Even more poorly understood are the by-products of mycotoxin degradation, particularly under the influence of strong oxidizing agents such as sodium hypochlorite and/or ozone, agents frequently used or misused by remediation personnel in the industry. More research is required in this field to better understand the relationship of fungal contamination, mycotoxin production on building substrates and building related disease.

Endotoxins

Endotoxin is the name given to a group of heat stable lipopolysaccharide molecules present in the cell walls of gram-negative bacteria that have a certain characteristic toxic effect. The lipid portion of each molecule is responsible for its toxicity and can vary between bacterial species and even from cell to cell. When inhaled, endotoxin creates an inflammatory response in humans that may result in fever, malaise, alterations in white blood cell counts, headache, respiratory distress and even death. It is common to the environment due to the ubiquitous nature of Gram-negative bacteria. Exposure to elevated levels of endotoxin primarily occurs through exposure to aerosols from specific reservoirs such as cotton mills, wastewater treatment facilities, air washers, humidifiers and any other occupational settings where Gram-negative bacteria can flourish.

Mycotoxins

In addition to their roles as irritants and allergens, many fungi produce toxic chemical constituents (Kendrick, 1992; Miller, 1992; Wyllie and Morehouse, 1977).

Samson and co-workers (1996) defined mycotoxins as "fungal secondary metabolites that in small concentrations are toxic to vertebrates and other animals when introduced via a natural route". These compounds are non-volatile and may be sequestered in spores and vegetative mycelium or secreted into the growth substrate. The mechanism of toxicity of many mycotoxins involves interference with various aspects of cell metabolism, producing neurotoxic, carcinogenic or teratogenic effects (Rylander, 1999). Other toxic fungal metabolites such as the cyclosporins exert potent and specific toxicity on the cellular immune system (Hawksworth et al., 1995); however, most mycotoxins are known to possess immunosuppressant properties that vary according to the compound (Flannigan and Miller, 1994). Indeed, the toxicity of certain fungal metabolites such as aflatoxin, ranks them among the most potently toxic, immunosuppressive and carcinogenic substances known (ibid.). There is unambiguous evidence that ingestion exposure as well as exposures by the inhalation pathway have been correlated with outbreaks of human and animal mycotoxicoses (Abdel-Hafez and Shoreit, 1985; Burg et al., 1982; Croft et al., 1986; Hintikka, 1978; Jarvis, 1986; Norbick et al., 1990; Sorenson et al., 1987; Schiefer, 1986). Several common mycotoxigenic indoor fungi and their respective toxins are listed.

Volatile Fungal Metabolites

During exponential growth, many fungi release low molecular weight, volatile organic compounds (VOCs) as products of secondary metabolism. These compounds comprise a great diversity of chemical structure, including ketones, aldehydes and alcohols as well as moderately to highly modified aromatics and aliphatics. Cultural studies of some common household moulds suggests that the composition of VOCs remains qualitatively stable over a range of growth media and conditions (Sunesson et al., 1995). Furthermore, the presence of certain marker compounds common to multiple species, such as 3-methylfuran, may be monitored as a proxy for the presence of a fungal amplifier (Sunesson et al., 1995). This method has been suggested as a means of monitoring fungal contamination in grain storage facilities (Bjesson et al., 1989; 1990; 1992; 1993).

Limited evidence suggests that exposure to low concentrations of VOCs may induce respiratory irritation independent of exposure to allergenic particulate (Koren et al., 1992). Volatile organic compounds may also arise through indirect metabolic effects. A well-known example of this is the fungal degradation of urea formaldehyde foam insulation.

Fungal colonization of this material results in the cleavage of urea from the polymer, presumably to serve as a carbon or nitrogen source for primary metabolism. During this process formaldehyde is evolved as a derivative, contributing to a decline in IAQ (Bissett, 1987).

The present study was conceived with two primary objectives. First, this investigation shall characterize the fungal biodiversity of house dust. This work shall investigate correlations between dustborne fungal species, and examine the ecological similarity of positively associated taxa based on the hypothesis that positively associated dustborne fungi are likely to share habitat characteristics.

From this, a second hypothesis follows that mechanisms that permit the entry or concentration a given species will tend to facilitate the entry of other positively correlated taxa. A second objective of this research is to assess the extent of genotypic variability in two dustborne *Penicillia*, *P. brevicompactum* and *P. chrysogenum*.

Mycotoxins of significance produced by indoor fungi

- Mycotoxin Primary health effect Fungal producers
- Aflatoxins Carcinogens, hepatotoxins: *Aspergillus flavus*, *A. parasiticus*
- Citrinin Nephrotoxin: *Penicillium citrinum*, *P. verrucosum*
- Cyclosporin Immunosuppressant: *Tolypocladium inflatum*
- Fumonisin, Carcinogens, neurotoxins: *Fusarium moniliforme*,
- *F. proliferatum*
- Ochratoxin A Carcinogen: *A. ochraceus*, *P. verrucosum*
- Patulin Protein synthesis inhibitor: *A. terreus*
- Nephrotoxin: *Paecilomyces variotii*
- *P. expansum*
- *P. griseofulvum*
- *P. roquefortii*
- Sterigmatocystin Carcinogen, hepatotoxin: *A. nidulans*