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Feasibility of assessing mycotoxin exposure in the indoor environment using wastewater-based epidemiology

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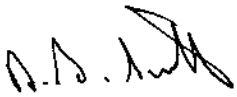

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1. EXECUTIVE SUMMARY

Many New Zealand homes are known to suffer from mould and dampness. This may pose a health hazard not only due to the potential for development of fungal infections, or mycoses, but also due to the production of toxic chemicals, known as mycotoxins, by some mould species.

Mycotoxin exposure in humans has mostly been associated with the consumption of contaminated foods. However, exposure can also occur through inhalation and dermal contact with mycotoxins present in the air or dusts of mould contaminated indoor environments. A variety of different mould species are known to grow in indoor environments and are particularly prevalent in damp or water-damaged buildings. This is of added concern in New Zealand due to the prevalence of mould and dampness in New Zealand homes and the 'leaky homes crisis' in which some buildings constructed during the 1990s to mid-2000s suffer from weathertightness issues, leading to water ingress and mould development.

A recent pilot study conducted by the Building Research Association of New Zealand (BRANZ) identified a range of mycotoxins in New Zealand homes. This study is currently being extended to a larger number of homes across New Zealand. Mycotoxin production by the black mould, *Stachybotrys chartarum*, in New Zealand leaky homes is also being investigated, with this species being the most common mould present in wet rots in water-damaged buildings in New Zealand.

Traditional methods for assessing human exposure to mycotoxins include dietary surveys, analysis of foodstuffs, and biomonitoring studies. More recently, wastewater-based epidemiology (WBE) has also been applied to assessment of human mycotoxin exposure. However, these methods have all primarily focused on dietary exposure.

In New Zealand, the Ministry for Primary Industries assesses the risk posed by dietary exposure to mycotoxins through the Mycotoxin Surveillance Program. However, no biomonitoring of mycotoxin exposure has been performed.

The aim of this report was to assess the feasibility of specifically assessing exposure to mycotoxins present in the indoor environment using WBE.

This feasibility assessment consisted of four main stages:

1. Review of published and grey literature to identify which mycotoxins have been found in the indoor environment.
2. High-level overview of the potential human health hazard posed by those mycotoxins most frequently identified in the indoor environment.
3. Review of which mycotoxins have been assessed internationally using urinary biomonitoring.
4. Assessment of the suitability of those mycotoxins most frequently identified in the indoor environment, and which pose a potential health hazard, for WBE.

The review of mycotoxins reported in indoor environments included almost thirty different studies and identified over 140 different fungal secondary metabolites present in indoor environment samples, which included building materials, surface swabs, floor dust and dust settled on above floor surfaces (eg, window sills, door frames; referred to as settled dust), and indoor air. Twenty-three of the most-frequently detected metabolites, all of which are known to be mycotoxins, were chosen for further assessment.

Information identified on the potential health risks posed by these twenty-three mycotoxins mostly focused on dietary exposure, with limited information identified in relation to inhalation or dermal exposure. Additionally, much of the information on potential health risks was solely based on animal studies, and for some of the emerging mycotoxins there was very little information available on the potential health risks. Based on the available information, twenty-two of the mycotoxins were deemed to pose a potential human health hazard and were taken forward for assessment of suitability for WBE.

An essential criterion when choosing a target for monitoring using WBE, referred to as a biomarker, is that it be excreted in urine and/or faeces. As such, the first step taken in assessing the feasibility of WBE for the twenty-two candidate mycotoxins was to identify whether they had already been a target of urinary biomonitoring internationally. Review of grey and published literature identified seventy-nine different studies ranging in size from 10 to 2,212 participants. Ten of the twenty-two candidate mycotoxins were found to have been assessed using urinary biomonitoring, but only eight were detected in any of the samples.

Further assessment of the candidate mycotoxins involved review of any additional information pertaining to their potential excretion in urine and/or faeces, including whether they are excreted unchanged or as metabolites. Their potential presence in foodstuffs was also considered to ascertain whether dietary exposure would also contribute to levels measured using WBE.

Based on these assessments, the following 10 mycotoxins were determined to be promising candidates for WBE:

- Sterigmatocystin
- Enniatins A, A1, B and B1
- Beauvericin
- *Alternaria* toxins alternariol and alternariol monomethyl ether
- Roquefortine C
- Griseofulvin

However, all these mycotoxins have also been identified in various foodstuffs, and as such there may be non-human sources contributing to their presence in wastewater and therefore levels detected using WBE (eg, mouldy food disposed of through kitchen sink food waste disposers). For some of these candidates a suitable human metabolite is available which overcomes this problem. However, preliminary work will be required to confirm the chosen biomarkers are stable and reliably detectable in wastewater.

Given all 10 of the promising candidates are also present in foodstuffs, specific assessment of indoor exposure would require estimation of baseline dietary exposure levels, which could then be subtracted from detected levels to estimate indoor exposure. This could be achieved using biomonitoring as has been conducted internationally. However, this may be complicated by factors such as cultural dietary preferences and seasonal differences in dietary mycotoxin exposure. New Zealand already has a urinary biomonitoring program to which mycotoxins could be added. However, based on animal studies, many of these candidate mycotoxins are preferentially excreted in faeces. As such, urinary biomonitoring may not give a true picture of exposure. As WBE measures both urinary and faecal excretion it should provide a more accurate estimation of exposure for those mycotoxins excreted in both urine and faeces.

Overall, this report has identified ten mycotoxins which are promising candidates for assessing the exposure to mycotoxins in the indoor environment using WBE. However, preliminary work will be required to determine whether the chosen biomarkers are stable and detectable in wastewater, and to establish baseline dietary exposure levels to allow for estimation of indoor exposure.

2. INTRODUCTION

Mycotoxins are secondary metabolites produced by a range of fungal species (Bennett & Klich 2003). These metabolites comprise a diverse group of more than 300 members grouped together on the basis that they can cause disease or death in humans and other vertebrates (Bennett & Klich 2003, Ji et al 2016). Not all fungal secondary metabolites are toxic and not all fungal species produce mycotoxins (Zain 2011). Some fungi are known to produce more than one different mycotoxin and the same mycotoxin may be produced by more than one fungal species (Zain 2011). Exposure to mycotoxins can lead to mycotoxicoses, which are distinct from fungal infections or mycoses, in that they are "poisoning by natural means" more analogous in pathology to exposure to chemicals such as heavy metals or pesticides (Bennett & Klich 2003).

2.1 PATHWAYS FOR EXPOSURE TO MYCOTOXINS

The majority of human mycotoxin exposures are thought to occur through consumption of contaminated food, but inhalation and dermal exposure are also important routes for exposure (Bennett & Klich 2003). The focus of this report is mycotoxin exposure in the indoor environment. However, this section will provide a brief overview of other exposure pathways including dietary and outdoor exposure. Occupational exposures are not specifically considered as these are outside of the scope of this report.

2.1.1 Dietary exposure

Mycotoxins have been identified in a range of different foodstuffs, leading to human exposure when these contaminated foods are consumed, or through consumption of animal products from animals fed contaminated feed (Gracia-Lor et al 2020, Zain 2011). These chemicals are generally highly stable and survive storage and processing (Rodríguez-Carrasco et al 2014). Additionally, most mycotoxins are stable at high temperatures so cannot be detoxified by thermal treatments (Gil-Serna et al 2019), and are generally not destroyed by normal cooking processes (Kumar et al 2017). Some of the most common mycotoxins identified in foods are summarised in Table 1.

In New Zealand, the risks posed by dietary exposure to mycotoxins is assessed by the Ministry for Primary Industries (MPI) Mycotoxin Surveillance Program[1]. Risk assessments have been conducted for a variety of mycotoxins, as summarised in Table 2.

The potential for contamination of drinking water with mycotoxins is also being investigated, given several studies have identified fungal genera with members known to produce mycotoxins in drinking water systems (reviewed by Afonso et al (2021)). Indeed, a recent investigation of a treated drinking-water distribution system in South Africa detected several mycotoxins, although the authors noted that "the estimated average daily dose (ADD) for detected mycotoxins was below the tolerable daily intake (TDI), suggesting no toxicological

[1] https://www.mpi.govt.nz/science/food-safety-and-suitability-research/food-science-research/chemical-hazard-and-mycotoxin-research/ Accessed 13 July 2022

risk" (Mhlongo et al 2020). Similarly, Afonso et al (2021) noted that "the concentration of mycotoxins in drinking water is likely to be very diluted and, for the time being, has not been identified as the source of symptoms attributable to mycotoxins".

2.1.2 Outdoor exposure

There are a variety of ways in which exposure to mycotoxins in the outdoor environment may occur, and this will be covered in a future report. As such, this section serves to provide a high-level overview of key routes for exposure, some of which are highlighted in Figure 1 using the *Fusarium* mycotoxin zearalenone as an example.

Several studies have highlighted the presence of mycotoxins in surface waters (Bucheli et al 2008, Gromadzka et al 2009), and they have been referred to as "emerging surface water contaminants" (Székács 2021). Exposure to mycotoxins present in surface waters may occur during recreational usage of contaminated waterways, or through consumption of contaminated mahinga kai.

The two main routes for mycotoxins to enter aquatic environments are runoff from contaminated agricultural fields and effluents from wastewater treatment plants (Schenzel et al 2010). Two *Fusarium* mycotoxins, zearalenone and deoxynivalenol, were identified in concentrations of up to 35 and 4,900 ng/L respectively in drainage from an artificially inoculated winter wheat field in Switzerland (Bucheli et al 2008). Similarly, water from drainage ditches in agricultural areas in Poland was found to contain up to 48 ng/L of the *Fusarium* mycotoxin fumonisin B1 (Waśkiewicz et al 2015). Mycotoxins have also been detected in a range of soils, and may leach into groundwaters (reviewed in Juraschek et al (2022)). Additionally, excrement from farm animals fed mycotoxin-contaminated feed may also add to surface water contamination via runoff from paddocks containing grazing animals or areas where contaminated manure has been applied (Schenzel et al 2012).

Several studies have identified wastewater treatment plant effluent as an important source of mycotoxins to aquatic environments (Kolpin et al 2014, Schenzel et al 2012, Schenzel et al 2010, Wettstein & Bucheli 2010). Much of the mycotoxin load present in wastewater is likely due to human excretion (Schenzel et al 2012). Indeed, this is the premise of two recent wastewater-based epidemiology (WBE) studies which assessed population exposure to mycotoxins based on the presence of selected mycotoxins or their metabolites in untreated wastewater (Berzina et al 2022, Gracia-Lor et al 2020). These chemicals are not always completely removed by wastewater treatment processes (Gromadzka et al 2015, Laganà et al 2004, Laganá et al 2001) leading to their presence in waterways receiving wastewater treatment plant discharges.

Table 1 Summary of the main mycotoxins found in food

Mycotoxin	Fungal (mould) species	Food commodity
Aflatoxins	*Aspergillus flavus* *A. parasiticus* *A. nomius*	Wheat, tree nuts, maize, cotton, peanuts, eggs, milk, meat, dried fruit, spices
Ochratoxin A	*A. ochraceus,* *A. carbonarius* *Penicillium verrucosum* *P. nordicum* *A. niger*	Coffee beans, oats, wheat, maize, wine, dried fruits, spices, eggs, meat
T-2/HT-2 toxin (type A trichothecenes)	*Fusarium sporotrichoides* *F. poae* *F. acuminatum* *F. equiseti* *F. langsethiae* *F. sibiricum*	Barley, oats, wheat, maize
Deoxynivalenol and related type B trichothecene mycotoxins	*F. graminearum* *F. culmorum*	Wheat, maize, oats, beer
Fumonisins	*F. fujikuroi* *F. proliferatum* *F. verticilloides* *A. niger*	Maize-based foods
Ergot alkaloids	*Claviceps* species	Barley, oats, rye, spelt, wheat
Patulin	*P. expansum* *P. carneum* *P. coprobium* *A. clavatus* *A. giganteus* *Byssochlamys nivea* *Paecilomyces saturatus*	Apples, grapes, plums, peaches, pears, tomatoes

Zearalenone	*F. graminearum,* *F. cerealis* *F. culmorum* *F. equiseti* *F. verticillioides*	Maize, barley, oats, sorghum, wheat, eggs, beer

Information from Janik et al (2020), Cressey and Pearson (2020) and Ashmore et al (2020).

Table 2 Reports prepared by the New Zealand Mycotoxin Surveillance Program

Report focus	Reference
Mycotoxins in the New Zealand food supply	Cressey et al (2006)
Aflatoxins in maize products	Cressey et al (2008)
Aflatoxins and ochratoxin A in dried fruit and spices	Cressey et al (2009)
Aflatoxins in nuts and nut products	Cressey et al (2010)
Dietary exposure to aflatoxins	Cressey and Reeve (2011)
Ochratoxin A in cereals, wine, beer and coffee	Cressey et al (2011)
Trichothecenes in cereal products	Cressey et al (2014)
Dietary exposure to ochratoxin A and trichothecenes	Cressey and Pearson (2014b)
Mycotoxins in the New Zealand food supply (update to 2006 report)	Cressey and Pearson (2014a)
Fumonisins in maize-based products and wine	Cressey et al (2020)
Dietary exposure to fumonisins	Cressey and Pearson (2020)
Ergot alkaloids in cereal-based foods and rye	Ashmore et al (2020)
Aflatoxins and ochratoxin A in New Zealand spices	Rowland et al (2021)

Reports available at https://www.mpi.govt.nz/science/food-safety-and-suitability-research/food-science-research/chemical-hazard-and-mycotoxin-research/

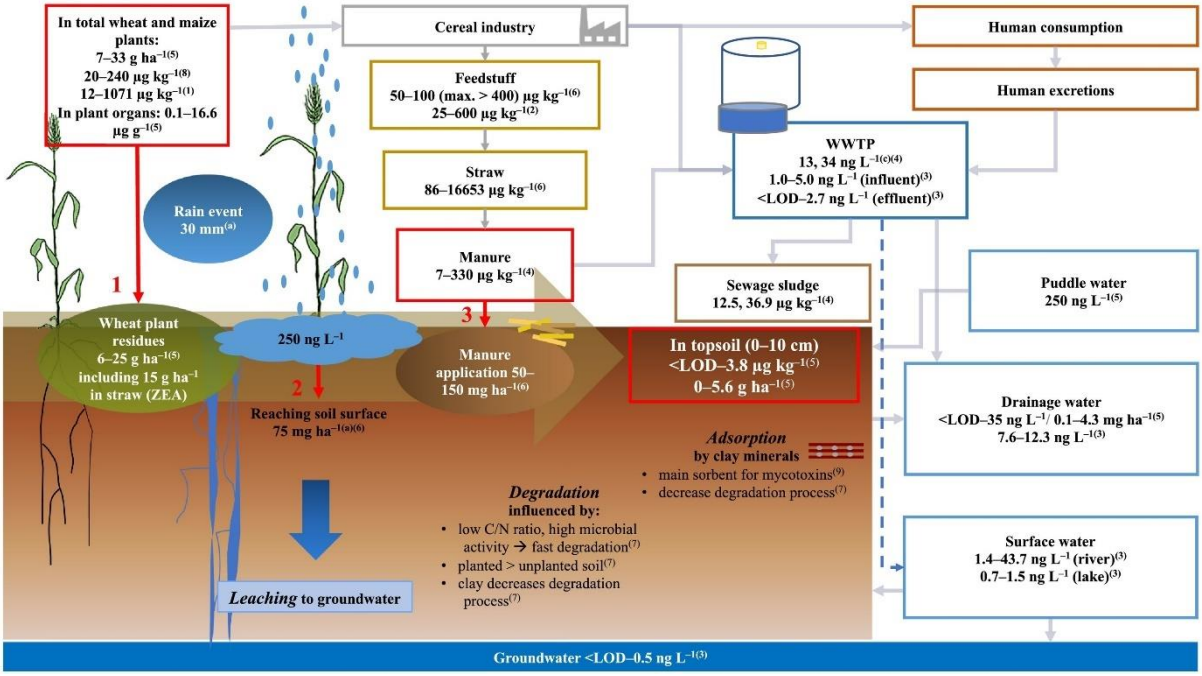


Figure 1 Possible environmental distribution of the *Fusarium* mycotoxin zearalenone

Reproduced from Juraschek et al (2022). LOD, limit of detection; WWTP, wastewater treatment plant; ZEA, zearalenone.

3. MYCOTOXIN EXPOSURE IN THE INDOOR ENVIRONMENT

People spend around 90% of their lives indoors, and are estimated to inhale approximately 15 m^3 of ambient air each day (Dyląg et al 2022). It has previously been noted that poor housing quality is consistently linked to poor health outcomes, with development of respiratory disorders positively correlated with living in cold, damp, mouldy housing (Clarke et al 2021). A national random telephone survey of 613 New Zealand households published in 2005 found that 35% had mould in one or more rooms (Howden-Chapman et al 2005). A more recent Statistics New Zealand Housing in Aotearoa 2020 report found that mould and dampness are common in New Zealand homes, with 1 in 6 having visible mould with a total area larger than A4 size (Stats NZ 2020). This report also highlighted that Māori and Pasifika are more likely to be living in homes affected by mould and dampness than other ethnicities (Stats NZ 2020). Additionally, cold, dampness and mould were reported to be more common in households that were non-owner-occupier, had four or more members, and had insufficient money for everyday needs (Stats NZ 2020).

A recent book published by the He Kāinga Oranga/Housing and Health Research Programme stated that "New Zealand is a country noted for its high level of dampness in housing, which probably reflects high outdoor humidity, relatively poor housing stock, inadequate insulation and inadequate heating" (Howden-Chapman et al 2009). Of additional concern is the 'leaky homes' crisis in which some buildings constructed between the 1990s and mid-2000s are affected by weathertightness issues (Clarke et al 2021, Yates 2003). In some cases, these issues have allowed moisture to ingress into the timber framing and wallboards, leading to widespread mould development (Clarke et al 2021).

A wide variety of different fungal (mould) species have been identified in buildings, with *Cladosporium*, *Penicillium, Aspergillus* and *Alternaria* species noted to be the some of the most common (reviewed in Bellanger et al (2009)). The black mould *Stachybotrys chartarum* also gained notoriety when it was associated with a cluster of acute idiopathic pulmonary haemorrhage cases in infants living in water-damaged homes in Ohio between 1993 and 1998 (Dearborn et al 1999). However, later expert review found there were "significant methodological shortcomings in the investigation, concluding that no causal link between mould infestation and the observed respiratory symptoms could be established" (discussed in Clarke et al (2021)). Despite this conclusion, there is still considerable public concern over 'toxic black mould' (Clarke et al 2021). A survey of wet rots in water-damaged buildings in New Zealand found that *S. chartarum* was the most commonly identified microbe, present in 46/59 building material samples tested (Howden-Chapman et al 2009).

A recent study by the Building Research Association of New Zealand (BRANZ) identified spores from several fungal species in air samples taken from 88 homes across New Zealand (Plagmann et al 2021) (Table 3). Excessive or elevated amounts of *Cladosporium* and *Penicillium*/*Aspergillus* spores were identified in houses in Auckland, Dunedin, Christchurch and Wellington. *Stachybotrys* and *Chaetomium* were also detected in some samples, which is generally "indicative of a prolonged leak or major wetting event" (Plagmann et al 2021).

Table 3 Results of airborne spore sampling in New Zealand houses

House ID	*Stachybotrys*	*Chaetomium*	*Penicillium/ Aspergillus*		*Cladosporium*		Bacterial clusters (high levels)	Other spore types
			Excessive	Elevated	Excessive	Elevated		
AK4.3	Bedroom 1							
DN1.5			Bedroom 1 Living room	Bathroom		Living room	Living room** Bedroom 1	Living room
AK1.1		Bathroom*	Bedroom 1	Bathroom Living room	Bedroom 1		Bedroom 1 Bathroom*** Living room	Bedroom 1 Living room
WN2.1			Bedroom 3		Bedroom 3		Bedroom 3	Bedroom 3
AK3.4			Bedroom				Bedroom	Bedroom
AK2.2			Bedroom 2				Bedroom 2	
WN3.1			Bedroom 3		Bedroom 3		Bedroom 3	
CH2.3				Bedroom 1		Bedroom 1	Bedroom 1	
AK1.3				Bedroom		Bedroom	Bedroom	Bedroom
AK4.1				*Bedroom**				Bedroom
CH1.3		Living room*		Bathroom*				Living room Bathroom
DN1.2		Bathroom*						

* Observed level only slightly raised – unlikely to result in health issues.
** Likely to be due to increased moisture resulting in bacterial growth.
*** Likely to be indicative of condensation and a lack of good ventilation.

Reproduced from Plagmann et al (2021). AK, Auckland; DN, Dunedin; WN, Wellington, CH, Christchurch.

Indoor mycotoxin exposure may also not only through inhalation but also via direct dermal/skin contact with mouldy surfaces or mycotoxins present in floor or surface dust, as some mycotoxins have been shown to penetrate skin *in vitro* (Boonen et al 2012, Taevernier et al 2016).

In Australia, a recent Government inquiry investigated biotoxin-related illness, particularly with respect to water-damaged buildings (House of Representatives Standing Committee on Health Aged Care and Sport 2018). The term biotoxins refers to “substances of biological origin, some of which can produce toxic effects in humans” and includes mycotoxins but also toxins produced by other organisms such as plants (House of Representatives Standing Committee on Health Aged Care and Sport 2018). The inquiry considered Chronic Inflammatory Response Syndrome (CIRS) – a range of health effects often associated with water-damaged buildings (House of Representatives Standing Committee on Health Aged Care and Sport 2018). The Committee recommended that there be research to “examine any links between mould and biotoxins and complex symptoms most commonly reported as typifying CIRS”. However, “Biotoxin-related illnesses and CIRS are not widely recognised medical conditions among the Australian medical profession” and “the Department of Health stated that, at this stage, ‘the scientific evidence is not sufficient … to accept the assertion that exposure to environmental biotoxins is causing [CIRS]” (House of Representatives Standing Committee on Health Aged Care and Sport 2018). Additionally, the Department of Health stated that “The department understands that some people suffer from a collection of chronic debilitating symptom complexes that have been attributed to exposure to mould … At this stage, there is insufficient evidence to support a direct link between these symptoms and mould exposure” (House of Representatives Standing Committee on Health Aged Care and Sport 2018). Indeed, CIRS does not appear to be formally recognised by the World Health Organization (WHO), although they do note that “the amount of water on or in

materials is the most important trigger of the growth of microorganisms, including fungi" and "microbial growth may result in greater numbers of spores, cell fragments, allergens, mycotoxins, endotoxins, β-glucans and volatile organic compounds in indoor air. The causative agents of adverse health effects have not been identified conclusively, but an excess level of any of these agents in the indoor environment is a potential health hazard" (WHO Regional Office for Europe 2009).

3.1 MYCOTOXINS IDENTIFIED IN THE INDOOR ENVIRONMENT

Numerous studies have assessed the presence of mycotoxins in samples taken from the indoor environment, particularly in buildings with moisture issues or water damage. Over 140 different fungal secondary metabolites were reported in the studies identified during preparation of this report, as summarised in Appendix Table 15. However, it is important to note that these analyses may be subject to matrix effects – "analyte signal enhancement or suppression caused by other components of the parent material the analyte was extracted from (the 'matrix') [eg, dust, building materials], which cause the instrument to over- or under-report the actual analyte concentration" (Clarke et al 2021). House dust, in particular, has been found to be particularly susceptible to matrix effects (Clarke et al 2021). The limit of detection will also vary depending on the technology employed (eg, thin layer chromatography (TLC) versus liquid chromatography tandem mass-spectrometry (LC-MS/MS)). As such, this report is less focused on the concentration or amount of mycotoxin identified in the different studies but rather what mycotoxins are most frequently identified in these studies. However, it is also important to note that when using mass spectrometry to identify contaminants, some compounds will ionize better than others, meaning they are detectable at lower levels, thereby biasing the frequency of detection of particular compounds (Peitzsch et al 2012). It is also important to note that although most of the compounds listed in Appendix Table 15 are mycotoxins, many of the studies simply assessed the presence of 'fungal secondary metabolites', and the toxicity of some of these metabolites is unknown.

Bearing these caveats in mind, Table 4 provides a summary of those mycotoxins identified in five or more studies. A brief overview of some of these studies is also provided below.

3.1.1 New Zealand studies

In New Zealand, BRANZ piloted a method which identified a variety of mycotoxins in carpet dust samples collected from across New Zealand (Plagmann et al 2021). Carpet dust samples were collected from the main bedroom of a residential dwelling by vacuuming a 1 m^2 area for 2 minutes and collecting the dust in a specialised nylon collection sock. Metabolite analysis was then performed by the Institute for Analytical Chemistry in Vienna, Austria.

The Health Research Council of New Zealand is funding a project investigating the potential health hazards associated with mycotoxins in residential dwellings. As part of this study, researchers from the University of Otago and BRANZ will examine the prevalence of mycotoxins in homes with either a recent history of water damage or large amounts of visible mould by comparing the levels in floor and surface dust samples taken from 100 leak/mould

affected homes and 100 control homes (Clarke et al 2021). The samples will initially be screened for over 300 microbial secondary metabolites using liquid chromatography-tandem mass spectrometry (LC-MS/MS), using methods described previously (Kirjavainen et al 2016). Levels of mycotoxins will be analysed and compared to lung function and reported respiratory health amongst occupants.

Table 4 Summary of the main mycotoxins identified in studies of the indoor environment

Toxin	Number of studies reporting*
Sterigmatocystin	19
Verrucarol	11
Enniatin B	10
Beauvericin	10
Satratoxin G	10
Satratoxin H	10
Enniatin A	9
Enniatin A1	9
Emodin	9
Enniatin B1	8
Alternariol monomethyl ether	8
Stachybotrylactam	8
Roquefortine C	8
Meleagrin	8
Alternariol	7
Griseofulvin	7
Trichodermol	6
Physcion	6
Chaetoglobosin A	6
Penicillic acid	5
3-Nitropropionic acid	5
Equisetin	5
Chanoclavine	5

*Where a study reports results from multiple countries, detections in each country are counted separately. Detections in different matrices in the same study are counted once. Results from artificial inoculations are not included. Compounds identified in the New Zealand BRANZ study of Plagmann et al (2021) are indicated in bold.

3.1.2 European studies

Several studies of mycotoxins in the indoor environment have been conducted in Germany. In a 2002 study, carpet dust samples taken from eight dwellings with visible mould growth and/or dampness were assessed for the presence of sterigmatocystin (Engelhart et al 2002). Samples from two of the dwellings had detectable amounts, although the concentrations were very low (2 – 4 ng/g of dust).

In a 2006 study, 15 samples of mouldy indoor materials (wallpaper, wall and gypsum board) were screened for the presence of eight macrocyclic trichothecene mycotoxins - roridin A, E and L-2, verrucarin A and J, and satratoxins F, G and H (Gottschalk et al 2006). Mycotoxins were detected in four of the samples, with satratoxins G and H present in all four samples at

concentrations ranging from 18 – 9,700 and 20 – 12,000 ng/cm^2 respectively (Gottschalk et al 2006). Roridin E and L-2, satratoxin F and verrucarin J were also detected but not quantified due to a lack of standards.

A follow-on study in 2008 screened air samples taken from the dwelling with the highest satratoxin levels (Gottschalk et al 2008). After a 15 hour sampling period over which 75 m^3 of air was filtered, a total of 19 ng of satratoxin G and 32 ng of satratoxin H was detected, corresponding to concentrations of 0.25 and 0.43 ng/m^3 respectively (Gottschalk et al 2008). A 2020 study specifically investigated the presence of *Stachybotrys* toxins in materials taken from water-damaged buildings (Jagels et al 2020). Detected toxins included satratoxins G and H, stachybotrylactam, and the recently discovered stachybotrychromenes (Jagels et al 2020).

The most recent German study, published in 2022, assessed the presence of 38 mycotoxins in dust samples taken from 21 households, with six displaying visible mould infestation (Lindemann et al 2022). Eight mycotoxins were identified – beauvericin, enniatins A, B and B1, sterigmatocystin, L-671,667, penitrem A and stachybonoid D (Lindemann et al 2022). The authors also compared LC-MS/MS with quadrupole time-of-flight (QTOF) high resolution mass spectrometry for identifying mycotoxins in highly matrix (dust)-loaded samples, and noted that the QTOF method allowed greater selectivity than LC-MS/MS, but was more sensitive to signal suppression by components present in the dust matrix (Lindemann et al 2022).

There have also been several studies of mycotoxins in the indoor environment in Finland. A 1997 study of indoor building materials from a children's day care centre which had suffered repeated water damage found satratoxins in the water-damaged gypsum liner (Andersson et al 1997). A study of 79 samples from mouldy interiors of buildings with moisture issues identified several mycotoxins, with at least one mycotoxin found in 34 (43%) of the samples (Tuomi et al 2000). The most prevalent mycotoxin was sterigmatocystin, present in 19 samples. Other detected mycotoxins included satratoxins G and H, citrinin and the trichothecenes deoxynivalenol, 3-acetyl-deoxynivalenol, diacetoxyscirpenol, T-2 tetraol and verrucarol (Tuomi et al 2000). The authors noted that although the detected fungi did not correlate well with the toxins identified, it is possible for different species to have been present in the same sample but to have proliferated at different stages (Tuomi et al 2000). For example, "a surface may be overgrown by *S. chartarum*, which prefers cellulosic matter with a high water content, with nitrogen deficiency promoting satratoxin production, but at an earlier stage of the water damage, at a lower relative humidity, *A. versicolor* could have dominated" (Tuomi et al 2000).

Another Finnish study, published in 2011, assessed the presence of non-volatile microbial secondary metabolites in settled floor dust from severely moisture damaged/damp houses (Vishwanath et al 2011). Several mycotoxins were detected including beauvericin, cytochalasin D, emodin, enniatins A, A1, B, B2, equisetin, sterigmatocystin, and the *Alternaria* toxins alternariol and alternariol monomethyl ether (Vishwanath et al 2011).

A second 2011 study assessed the presence of 186 different microbial secondary metabolites in 69 samples taken from severely moisture damaged homes (Täubel et al 2011). Sampled materials included indoor building materials, floor dust, dust from the residents vacuum cleaner bags and settled dust from surfaces above floor level. Of the 69

samples, all were positive for at least one of the analytes tested. Twenty-eight different fungal metabolites were detected including emodin, enniatins A, A1, B, beauvericin, equisetin, physcion, sterigmatocystin, meleagrin, chaetoglobosin A, stachybotrylactam, alternariol and alternariol monomethyl ether, roquefortine C, satratoxins G and H, ochratoxin A, and altenuene (full list in Appendix Table 15).

Another study, published in 2015, assessed the presence of 333 different fungal and bacterial secondary metabolites in living room floor dust samples collected from 93 homes of one-year old children (Kirjavainen et al 2016). Thirty-eight different fungal metabolites were detected, with brevianamide F, moniliformin, emodin, enniatins B and B1 and 3-nitropropionic acid detected in all 93 homes, and enniatins A and A1 in 92 of the homes (Kirjavainen et al 2016). The other fungal metabolites detected are summarised in Appendix Table 15, and the full list of metabolites screened for is provided in Appendix Table 16. The authors noted that "the total load of metabolites in living room floor dust (ng/m^2) tended to be increased if there was moisture damage in the living room or mould odour anywhere indoors during the site visit. The total number of different metabolites tended to also be higher in these cases, especially the number of metabolites present at loads higher than 0.1 ng/m^2" (Kirjavainen et al 2016).

The presence of mycotoxins in Finnish school buildings has also been investigated, with a 2012 study assessing the presence of over 180 different microbial secondary metabolites in primary school buildings in Finland (n = 59), Spain (n = 85) and the Netherlands (n = 92) as part of the Health effects of Indoor pollutants: integrating microbial, Toxicological and Epidemiological Approaches (HITEA) project (Peitzsch et al 2012). This study identified a variety of mycotoxins in settled dust and surface swab samples from buildings with and without moisture damage/dampness (Peitzsch et al 2012). The full list of analytes assessed is provided in Appendix Table 17. The authors noted that "settled dust derived from moisture damaged, damp schools contained larger numbers of microbial secondary metabolites at higher levels compared to respective dust samples from schools not affected by moisture damage and dampness" (Peitzsch et al 2012). Mycotoxins detected in dust samples from all three countries were beauvericin, emodin, enniatins A1, B, and B1, griseofulvin, meleagrin, penicillic acid, physcion, trichodermol and verrucarol (Peitzsch et al 2012). The surface swab samples were collected "exclusively from surfaces with visible or suspected microbial growth (eg, from locations with visible moisture damage)", and "generally, mycotoxin concentrations in mouldy spot surface swabs were clearly higher compared to settled dust samples, typically at least by one order of magnitude" (Peitzsch et al 2012). Emodin and meleagrin were detected in swab samples from schools in all three countries (Peitzsch et al 2012).

The presence of mycotoxins in Finnish school ventilation systems has also been investigated (Hintikka et al 2009). Dust samples were taken by swabbing surfaces of the ventilation system supply and exhaust air handling units and ductworks. Several mycotoxins were detected including beauvericin, penicillic acid, sterigmatocystin, chaetoglobosin A, gliotoxin, trichodermol, verrucarol, aflatoxin B1, satratoxin G/H and enniatins (Hintikka et al 2009).

The presence of mycotoxins in the indoor environment has also been assessed in other European countries including Belgium (Polizzi et al 2009), Denmark (Došen et al 2016, Nielsen et al 1999), France (Charpin-Kadouch et al 2006), Croatia (Jakšić et al 2021), Sweden (Bloom et al 2007, Bloom et al 2009b), Slovakia and Austria (Vishwanath et al

2009). In the Belgian study, the authors used LC-MS/MS to quantify the presence of 20 mycotoxins in 99 samples taken from seven water-damaged buildings (Polizzi et al 2009). They then extended this analysis by screening 23 of the 99 samples using liquid chromatography quadrupole time-of-flight mass spectrometry (LC-QTOF-MS), tentatively identifying 42 fungal metabolites, listed in Appendix Table 18.

3.1.3 Other studies

Several studies of mycotoxins present in water-damaged/mouldy buildings have also been conducted in the United States. The earliest study identified, published in 1986, assessed the presence of mycotoxins in mouldy building materials from a house in Chicago, Illinois infested with *S. chartarum* (referred to in the article by its former name *S. atra*) (Croft et al 1986). Detected mycotoxins included verrucarol (in hydrolysed extracts), verrucarins B and J, satratoxin H and trichoverrins A and B (Croft et al 1986).

In 2005, the presence of airborne macrocyclic trichothecenes in eight buildings in Texas with known *S. chartarum* contamination was investigated, with samples taken under static and disturbed air conditions (Brasel et al 2005). A specialised sampler modified to be able to separate and collect particulates smaller than conidia was also employed (Brasel et al 2005). Detected trichothecene concentrations ranged from <10 to >1,300 pg/m^3 of sampled air, which was significantly higher than in control buildings ($P < 0.001$) (Brasel et al 2005). Using the specialised sampler, trichothecenes were identified on particles smaller than conidia at concentrations of 1.8 – 63 trichothecene equivalents/m^3 of sampled air (Brasel et al 2005).

A separate study assessed the presence of macrocyclic trichothecenes in floor dust samples from two water-damaged buildings in the north-eastern United States (Saito et al 2016). Mycotoxins were extracted using methanol, then hydrolysed to convert the macrocyclic trichothecenes to verrucarol. The amount of verrucarol was then quantified, with levels up to 37 ng/g dust in some samples. However, the authors noted there were significant matrix effects which resulted in enhancement of detection by GC-MS/MS, leading to inaccurate measurement of true verrucarol levels (Saito et al 2016).

Dust collected from homes in New Orleans flooded during hurricane Katrina was also found to contain mycotoxins (Bloom et al 2009a). Sterigmatocystin was detected in two of the five houses tested, and verrucarol was detected in hydrolysed dust extracts from three houses, indicative of the presence of macrocyclic trichothecenes (Bloom et al 2009a).

A 2017 study assessed the ability of *P. brevicompactum, A. versicolor* and *S. chartarum* to produce mycotoxins on artificially-inoculated wallpaper, and assessed the ability of these toxins to be aerosolised (Aleksic et al 2017). Sterigmatocystin, mycophenolic acid and the macrocyclic trichothecenes roridin RL2, verrucarin J, and satratoxins G and H could all be produced on wallpaper, and some were aerosolised with air velocities “encountered under real-life conditions in buildings” (Aleksic et al 2017). The authors noted that although most of the mycotoxins were associated with particles ≥ 3 µm (which may correspond to spores), the macrocyclic trichothecenes, particularly satratoxin H and verrucarin J, could be found on smaller particles which are able to deeply penetrate the respiratory tract (Aleksic et al 2017). Various other mycotoxins have also been shown to be produced on artificially inoculated building materials, including alternariol, alternariol monomethyl ether, chaetoglobosins A and C, chrysogine, verrucofortine, verrucosidin, 3-methyoxy-viridicatin, naphtho-*γ*-pyrones,

nigragillin, orlandin, stachybotrylactam, stachybotrychromenes A, B and C, stachybotrysins B and C, stachybotrydial and stachybonoid D (full list in Appendix Table 15) (Gutarowska et al 2010, Jagels et al 2020, Nielsen et al 1999).

4. POTENTIAL HEALTH EFFECTS OF THE MAIN MYCOTOXINS FOUND INDOORS

Over 140 different fungal secondary metabolites were detected in studies of the indoor environment identified during preparation of this report (Appendix Table 15). Given it is impractical to provide a summary of the potential health risks posed by all these compounds, this section will focus on those mycotoxins most frequently identified in the indoor environment (as listed in Table 4), as these will likely be the most suitable candidates for assessment using wastewater-based epidemiology.

Most studies of the health effects of mycotoxin exposure have focused on oral exposure. As such, much of the information presented in this section is derived from dietary risk assessments. The health effects of mycotoxin exposure may be acute due to exposure to high levels, or chronic, where there is exposure over extended periods of time (Gil-Serna et al 2019).

In the following sections, information specific to those mycotoxins most frequently identified in studies of the indoor environment will be presented. It is important to note that for many of these toxins the information on potential health effects is limited. Additionally, this section is not intended to be a comprehensive description of all the known, or proposed, health effects of exposure to these mycotoxins, but rather is aimed at providing insight into whether they are likely to pose a human health risk and are therefore worthwhile considering for monitoring using WBE.

4.1 STERIGMATOCYSTIN

Sterigmatocystin is a polyketide-derived mycotoxin produced by over a dozen different *Aspergillus* species, as well as some other fungal species (JECFA 2017). It is structurally similar to the highly toxic aflatoxins and is an intermediate in the aflatoxin biosynthetic pathway (Figure 2). Some *A. nidulans* and *A. versicolor* strains are unable to convert sterigmatocystin into the aflatoxin precursor *O*-methyl sterigmatocystin so produce sterigmatocystin as a final product (Cressey & Pearson 2014a). The WHO considers sterigmatocystin to be both genotoxic and carcinogenic, and as such there is no tolerable daily limit[2] (JECFA 2017).

[2] https://apps.who.int/food-additives-contaminants-jecfa-database/Home/Chemical/6458 Accessed 25 August 2022

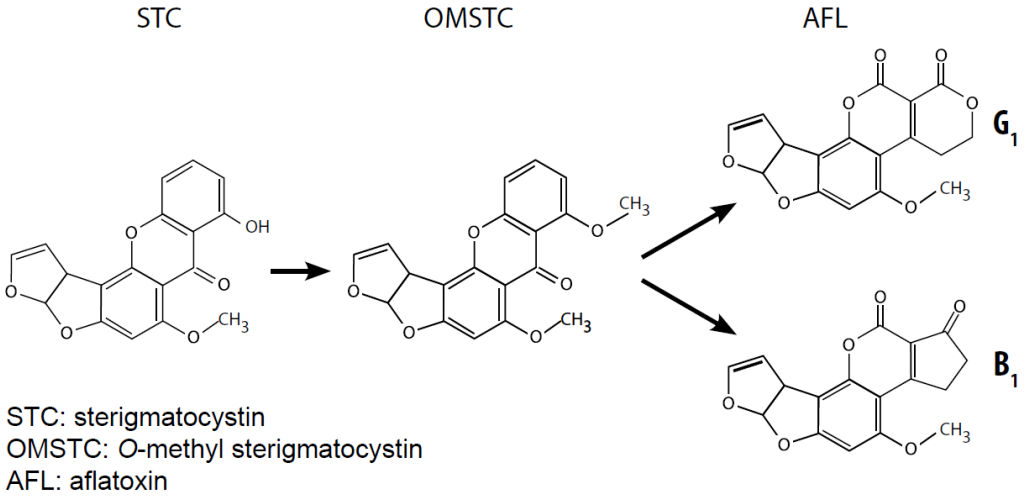


Figure 2 Biosynthesis of aflatoxins from sterigmatocystin

Reproduced from JECFA (2017).

A recent study assessed the toxicity of sterigmatocystin in rat lungs by intratracheal instillation (direct deposition of the toxin rather than via inhalation) (Jakšić et al 2020). The doses of sterigmatocystin tested, which were based on concentrations detected in dust from damp indoor areas, resulted in DNA damage (genotoxicity) but not significant cytotoxicity (Jakšić et al 2020). A separate study of the cytotoxicity of sterigmatocystin noted that it displayed 80-fold higher toxicity against A549 lung cells in comparison to Hep-G2 liver cells (Bünger et al 2004).

4.2 ENNIATINS AND BEAUVERICIN

The enniatins and beauvericin are structurally related cyclic hexadepsipeptide mycotoxins mainly produced by *Fusarium* species (EFSA 2014). As of 2014, twenty-nine naturally occurring enniatin analogues had been identified (EFSA 2014). The structures of beauvericin and the four enniatins frequently identified in studies of the indoor environment are shown in Figure 3.

In 2014, the European Food Safety Authority (EFSA) Panel on Contaminants in the Food Chain (CONTAM) released a “Scientific Opinion on the risks to human and animal health related to the presence of beauvericin and enniatins in food and feed” (EFSA 2014). This assessment considered the sum of enniatins A, A1, B and B1, and noted that “beauvericin and enniatins are cytotoxic” but “there are no reports on adverse effects in humans caused by food contaminated with beauvericin and enniatins” (EFSA 2014). The report concluded that “the large margins obtained for acute exposure to beauvericin and enniatins do not indicate concern for human health” (EFSA 2014). However, they do note that there may be “concern with respect to chronic exposure but no firm conclusion could be drawn” and “relevant *in vivo* toxicity data are needed to perform a human risk assessment” (EFSA 2014). Based on the lack of relevant toxicity data, the EFSA CONTAM panel applied the Threshold

of Toxicological Concern (TTC) approach, which assesses the "probability of adverse health effects and possible human health risks", and classifies the compound into one of three classes (I, II or III) using the Cramer decision tree (EFSA 2014). Using this approach beauvericin and the four enniatins were classified into group III which corresponds to a TTC of 1.5 µg/kg bw/day. This value was adopted for the enniatins based on no evidence of genotoxicity, but was reduced to 0.0025 µg/kg bw/day for beauvericin due to some *in vitro* studies suggesting potential genotoxic effects (EFSA 2014).

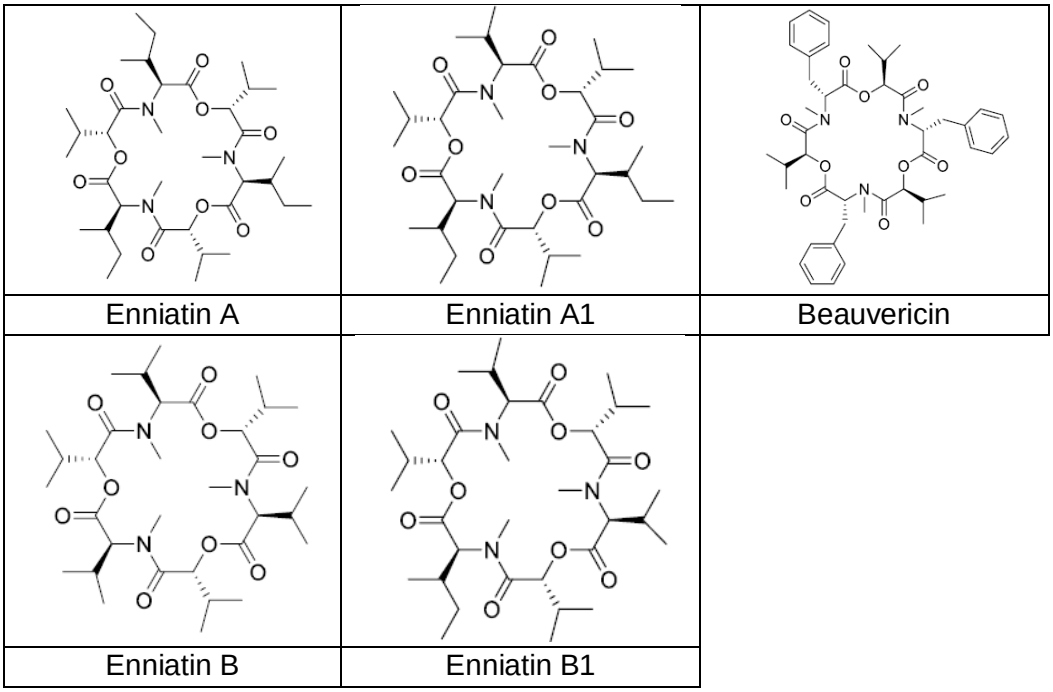


Figure 3 Structures of beauvericin and enniatins frequently present in indoor environments

Structures reproduced from Lindemann et al (2022).

Taevernier et al (2016) estimated a TDI of 5 µg/kg bw/day for enniatins based on a no observed adverse effect level (NOAEL) of 5 mg/kg bw. However, they note that "more (reliable) *in vivo* toxicity data, leading to a proper hazard characterisation are still urgently needed" (Taevernier et al 2016). More recently, Stanciu et al (2017) proposed a hypothetical value of 1 µg/kg bw/day for the sum of enniatins based on the TTC set by EFSA and provisional maximum tolerable daily intakes (PMTDIs) set for other mycotoxins.

Fusafungine, an antibiotic and anti-inflammatory nasal/oromucosal spray containing enniatins has been used as a human treatment for upper respiratory infections (EFSA 2014). However, the European Union[3] and several countries including Armenia, Azerbaijan, and the United Arab Emirates have withdrawn or suspended usage of fusafungine-containing medicines citing "increased risk of allergic reaction" and "limited evidence of benefit" (WHO 2020). Both beauvericin and enniatin B have been found to display potent cytotoxicity

[3] https://www.ema.europa.eu/en/medicines/human/referrals/fusafungine-containing-medicinal-products-oromucosal-nasal-use Accessed 25 August 2022

towards Chinese hamster V79 lung fibroblasts, which serve “as an *in vitro* surrogate for lung cells” (Behm et al 2012).

4.3 TRICHOTHECENES

There are more than 60 different trichothecene mycotoxins produced by several different fungal genera including *Stachybotrys*, *Fusarium*, *Trichoderma, Myrothecium, Trichothecium* and *Phomopsis* (Bennett & Klich 2003). These toxins can be classified as macrocyclic or non-macrocyclic (simple) based on whether there is a macrocyclic ester or an ester-ester bridge between C-4 and C-15 (Bennett & Klich 2003). Macrocyclic trichothecenes have been referred to as “some of the most potent mycotoxins yet discovered” (Clarke et al 2021). The structures of those trichothecenes most frequently identified in studies of the indoor environment are shown in Figure 4.

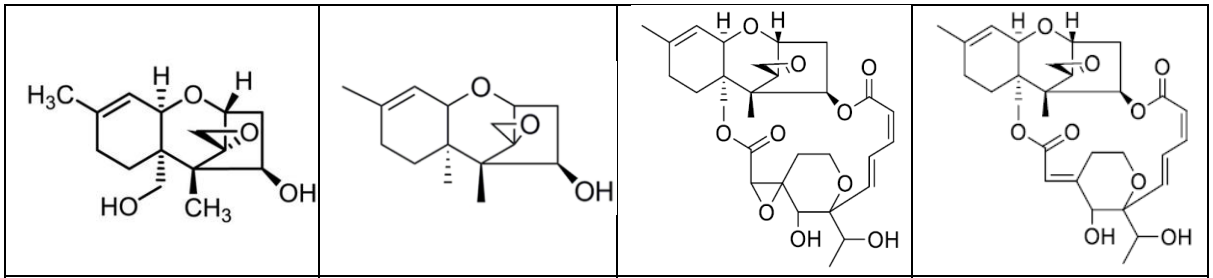


Figure 4 Structures of trichothecene mycotoxins frequently identified in the indoor environment

Reproduced from Lindemann et al (2022), Liu et al (2018) and Matsumoto et al (2017).

A recent publication by Clarke et al (2021) highlighted a very important point with respect to the toxicity of inhaled macrocyclic trichothecenes, which is that although most are “rapidly absorbed, distributed and metabolised, they tend to concentrate in specific cell types such as alveolar macrophages, and as the concentration of toxin at the site of deposition in the lungs will be greater locally than systemically, the environmental exposure required to cause local cellular injury will be lower than that required to achieve acute systemic toxicity”.

4.3.1 Verrucarol

Verrucarol is a simple trichothecene which can be formed by hydrolysis of macrocyclic trichothecenes (Bloom et al 2007), and thus can be used to screen hydrolysed extracts for total macrocyclic trichothecene presence (Clarke et al 2021, Peitzsch et al 2012). Using thin layer chromatography (TLC), verrucarol has been identified in extracts from some *S. chartarum* isolates (El-Kady & Moubasher 1982, Jarvis et al 2000). However, two other studies did not detect verrucarol production by *S. chartarum* (Bata et al 1985, Jarvis et al 1995).

Studies on the toxicity of verrucarol are limited. It has been noted to be structurally similar to the T-2 and HT-2 toxins but less toxic (Barel et al 1994). A report from 1969 found that verrucarol was cytotoxic to HEp2 cells at a concentration of 7.5 µg/mL and baby hamster kidney (BHK) cells at 5 µg/mL (Grove & Mortimer 1969). Trichothecenes exert their cytotoxicity through inhibition of cellular protein synthesis.

No assessments specifically addressing inhalation exposure to verrucarol were identified during preparation of this report.

4.3.2 Trichodermol

Trichodermol, also known as roridin C (Ueno & Ueno 1978), is a sesquiterpene mycotoxin derived from farnesyl diphosphate and is the immediate precursor of the trichothecene mycotoxin trichodermin (Figure 5) (Liu et al 2018). It can be formed by hydrolysis of trichodermin (Bloom et al 2007), but is also formed as a natural product by some *Trichoderma* species including *T. polysporum* and *T. sporulosum* (Adams & Hanson 1972) and *S. chartarum* (Jarvis et al 2000).

Similar to verrucarol, few studies of the toxicity of trichodermol were identified during preparation of this report. A book published in 1983 reported that trichodermol has an LD_{50} (dose that kills 50% of the test subjects) in mice of 500 – 1000 mg/kg, and is “about 100 times less toxic than the other trichothecenes” (Ueno 1983). This book also reported that trichodermol displays cytotoxicity to HeLa and HEp2 cells at 5.0 and 0.25 µg/ml respectively, due to inhibition of the protein synthesis elongation-termination step (Ueno 1983).

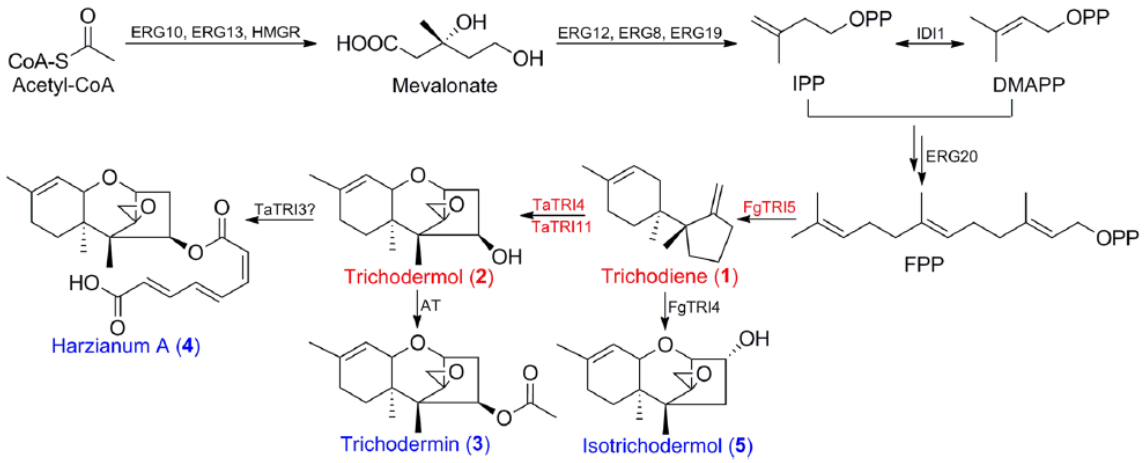


Figure 5 Biosynthesis of trichodermol

Reproduced from Liu et al (2018).

No assessments specifically addressing inhalation exposure to trichodermol were identified during preparation of this report.

4.3.3 Satratoxins G and H

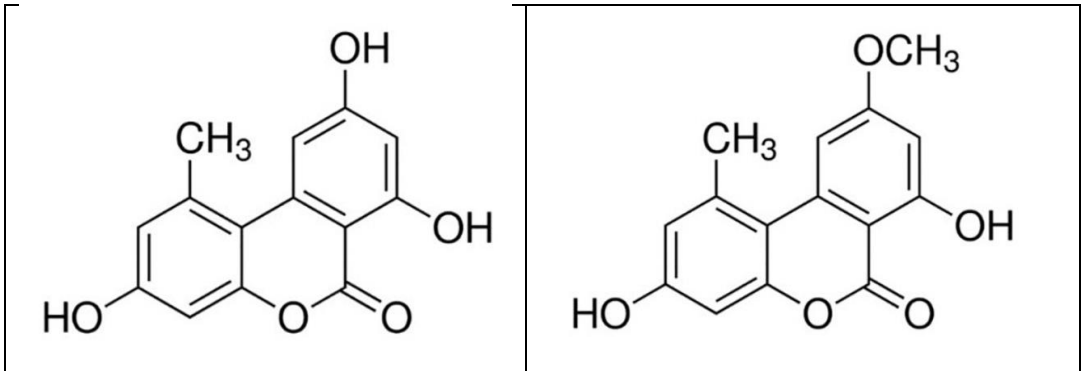
Satratoxins G and H are macrocyclic trichothecenes most notably produced by the toxic black mould, *S. chartarum*. These toxins are known to be “translational inhibitors that initiate both inflammatory gene expression and apoptosis *in vitro* following upstream activation of mitogen-activated protein kinases” (Carey et al 2012).

A 1986 mouse study found that intraperitoneal injection of satratoxins G and H resulted in LD_{50} values of 1.2 and 5.7 mg/kg body weight, respectively, and “both toxins caused extensive ulceration of the small intestine and a relatively mild damage of the lymphoid and hematopoietic tissues in lethal animals” (Yoshizawa et al 1986). Satratoxin G also caused “vacuolar and fatty degeneration of the hepatic cells” (Yoshizawa et al 1986). A report from 1983 also noted that satratoxins G and H were found in bedding straw that was responsible for the deaths of over 100 sheep in Hungary (Harrach et al 1983).

An intranasal instillation study assessing the toxicity of inhaled satratoxin G in mice found it caused inflammation in the nose and brain, and induced apoptosis of the olfactory sensory neurons, with severity increasing with increased dosage (Islam et al 2006). The authors noted that a single large dose (500 µg/kg bw) or five consecutive days of a lower dose (100 µg/kg bw) resulted in similar effects, suggesting the effects are cumulative (Islam et al 2006). Similar results were seen in an intranasal instillation study in rhesus monkeys, who are noted to have nasal airways that closely resemble those of humans (Carey et al 2012). The monkeys received either a single high dose (20 µg) or a lower dose (5 µg) over four consecutive days. Both groups developed acute rhinitis, apoptosis of the olfactory sensory neurons and atrophy of the olfactory epithelium (Carey et al 2012).

4.4 ALTERNARIOL AND ALTERNARIOL MONOMETHYL ETHER

Alternariol and alternariol monomethyl ether (sometimes referred to as just alternariol methyl ether) are dibenzo-α-pyrone mycotoxins produced by *Alternaria* species (EFSA 2011), including *A. alternata* and *A. tenuissima*, which have been identified in water-damaged buildings (Nielsen et al 1999). The structures of these toxins are shown in Figure 6.



Alternariol	Alternariol monomethyl ether

Figure 6 Structures of alternariol and alternariol monomethyl ether

Adapted from den Hollander et al (2022).

These toxins have been referred to as “emerging” mycotoxins (den Hollander et al 2022), and were considered by EFSA with respect to the risk they pose to animal and public health due to their presence in feed and food in 2011 (EFSA 2011). This Scientific Opinion noted that both alternariol and alternariol monomethyl ether are genotoxic *in vitro* but there was insufficient information on their toxicology to conduct a proper risk assessment (EFSA 2011). Instead they assigned a TTC of 2.5 ng/kg bw per day for both alternariol and alternariol monomethyl ether (EFSA 2011). In 2022, den Hollander et al. further noted that “thorough risk assessment [of alternariol and alternariol monomethyl ether] is mostly lacking” and “no legal maximum or guidance levels have been established yet for these emerging mycotoxins”. They showed that both alternariol and alternariol monomethyl ether are cytotoxic to human HepG2 and Caco-2 cell lines *in vitro* (den Hollander et al 2022).

No assessments specifically addressing inhalation exposure to alternariol or alternariol monomethyl ether were identified during preparation of this report. In their 2011 report, EFSA noted that “*Alternaria* spores are known to be allergenic via inhalation and *Alternaria* skin and nasal infections can occur in immunocompromised patients. However, since no evidence exists that these pathologies are related to *Alternaria* toxins, these toxicological endpoints are not considered in this opinion” (EFSA 2011).

4.5 OTHER MYCOTOXINS

The structures of the other mycotoxins most frequently identified in studies of the indoor environment are presented in Figure 7.

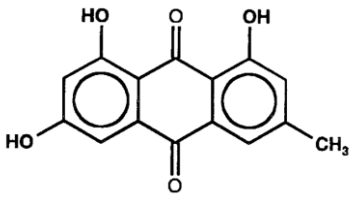
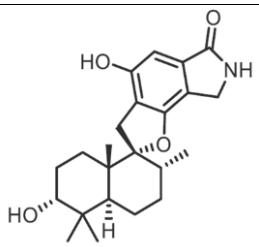
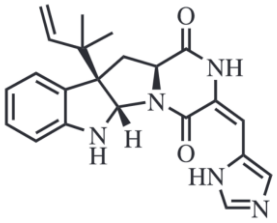
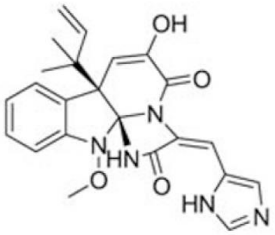
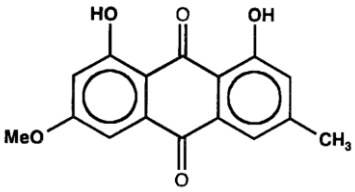
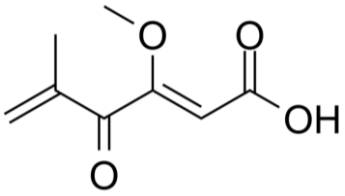
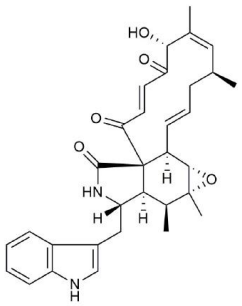
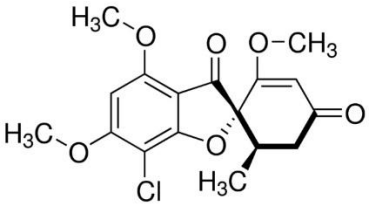
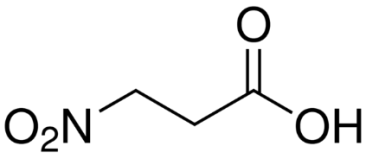
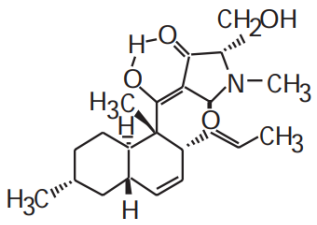
4.5.1 Emodin

Emodin is an anthraquinone produced by many plant and fungal species (Izhaki 2002, Masi & Evidente 2020, NTP 2001), and is known to be present in many widely used Chinese medicinal herbs (Dong et al 2016). A 2016 review noted that “emerging evidence indicates that emodin possesses a wide spectrum of pharmacological properties, including anticancer, hepatoprotective, anti-inflammatory, antioxidant and antimicrobial activities. However, emodin could also lead to hepatotoxicity, kidney toxicity and reproductive toxicity, particularly in high doses and with long-term use” (Dong et al 2016). Indeed, emodin has been shown to be genotoxic to mouse cells (Mueller et al 1999, Müller et al 1996), and results of a 2011 study suggest it can cause testicular toxicity in mice (Oshida et al 2011). A 2018 EFSA report on “Safety of hydroxyanthracene derivatives for use in food” noted that emodin has shown evidence of genotoxicity *in vitro* (EFSA ANS Panel et al 2018). Emodin has also been assessed by the United States Department of Health and Human Services National Toxicology Program[4] (NTP 2001). Based on results of a two-year feeding study, this assessment found no evidence of carcinogenic activity in male rats or female mice, and equivocal evidence of carcinogenic activity in female rats and male mice (NTP 2001).

[4] https://ntp.niehs.nih.gov/whatwestudy/testpgm/status/ts-518821.html?utm_source=direct&utm_medium=prod&utm_campaign=ntpgolinks&utm_term=ts-518821 Accessed 2 September 2022

However, emodin exposure had impacts on both rat and mouse kidneys (NTP 2001). The exact human health hazard posed by emodin exposure remains unclear, particularly given a separate mouse study reported it to be safe up to 80 mg/kg (Sougiannis et al 2021).

Figure 7 Structures of the other mycotoxins frequently identified in the indoor environment

		
Emodin Mueller et al (1999)	Stachybotrylactam Jagels et al (2020)	Roquefortine C Richard et al (2004)
		
Meleagrin Du et al (2010)	Physcion Mueller et al (1999)	Penicillic acid[5]
		
Chaetoglobosin A Perlatti et al (2020)	Griseofulvin[6]	3-Nitropropionic acid[7]
		
Equisetin Hazuda et al (1999)		

[5] https://www.medchemexpress.com/penicillic-acid.html Accessed 7 September 2022
[6] https://www.sigmaaldrich.com/NZ/en/product/sial/phr1534 Accessed 7 September 2022
[7] https://www.sigmaaldrich.com/NZ/en/product/sigma/n5636 Accessed 7 September 2022

Numerous studies have assessed the effect of emodin on lungs in terms of its protective role against disorders such as lung cancer (Su et al 2017) and silica-induced lung fibrosis (Yang et al 2016). However, no studies assessing potential toxicity of emodin due to inhalation were identified during preparation of this report.

4.5.2 Stachybotrylactam

Stachybotrylactam, also known as spirodihydrobenzofuranlactam 1 (Roggo et al 1996), is a spirocyclic drimane, or phenylspirodrimane, produced by *Stachybotrys* species (Došen et al 2016, Ulrich et al 2021).

Very little information on the potential toxicity of stachybotrylactam was identified during preparation of this report. However, phenylspirodrimanes have been noted to be immune- and neuro-toxic, and to inhibit the complement system (Ulrich et al 2021). Stachybotrylactam has been shown to inhibit the HIV-1 protease (Roggo et al 1996) and has also been reported to have immunosuppressive activity (discussed by Gutarowska et al (2010)).

No assessments specifically addressing inhalation exposure to stachybotrylactam were identified during preparation of this report.

4.5.3 Roquefortine C

Roquefortine C, or simply roquefortine (Ohmomo et al 1977), is an indole alkaloid mycotoxin produced by at least 30 *Penicillium* species, including *P. roqueforti* (Martín & Liras 2016), which is used as a starter culture for blue cheese production (Bünger et al 2004). Roquefortine C has been found at low levels in blue cheese but is “not thought to pose a significant health risk to consumers” as it is “very unstable in cheese” (Abbas & Dobson 2011).

There have been several cases of roquefortine poisoning reported in dogs, with some cases being fatal and initially thought to be due to strychnine poisoning due to similar clinical symptoms (Lowes et al 1992). A book published in 1978 reported an LD_{50} of 15 – 20 mg/kg for roquefortine intraperitoneally injected into rats (Ueno & Ueno 1978). Another study published the same year reported an average LD_{50} of 182 mg/kg for roquefortine intraperitoneally injected into mice (Arnold et al 1978). A test of the cytotoxicity of roquefortine C against human and mouse cell lines found no evidence of cytotoxicity “up to the limit of solubility” but noted that “the lack of apparent toxicity may be due to low bioavailability” (Bünger et al 2004). A review by Frisvad et al (2004) noted that roquefortine C is neurotoxic. However, it has also been noted that “different toxicity studies revealed that roquefortine C is not significantly toxic for humans and other mammals” (Martín & Liras 2016).

A 2005 study assessed the inflammatory and cytotoxic response of mouse lungs to intratracheal instillation of roquefortine C and found that it induced a significant inflammatory response but not cytotoxicity (Rand et al 2005). It was also noted that high doses of roquefortine C resulted in trembling and lethargy that resolved after 24 hours (Rand et al 2005).

4.5.4 Meleagrin

Meleagrin is an indole alkaloid mycotoxin formed from roquefortine C, as shown in Figure 8.

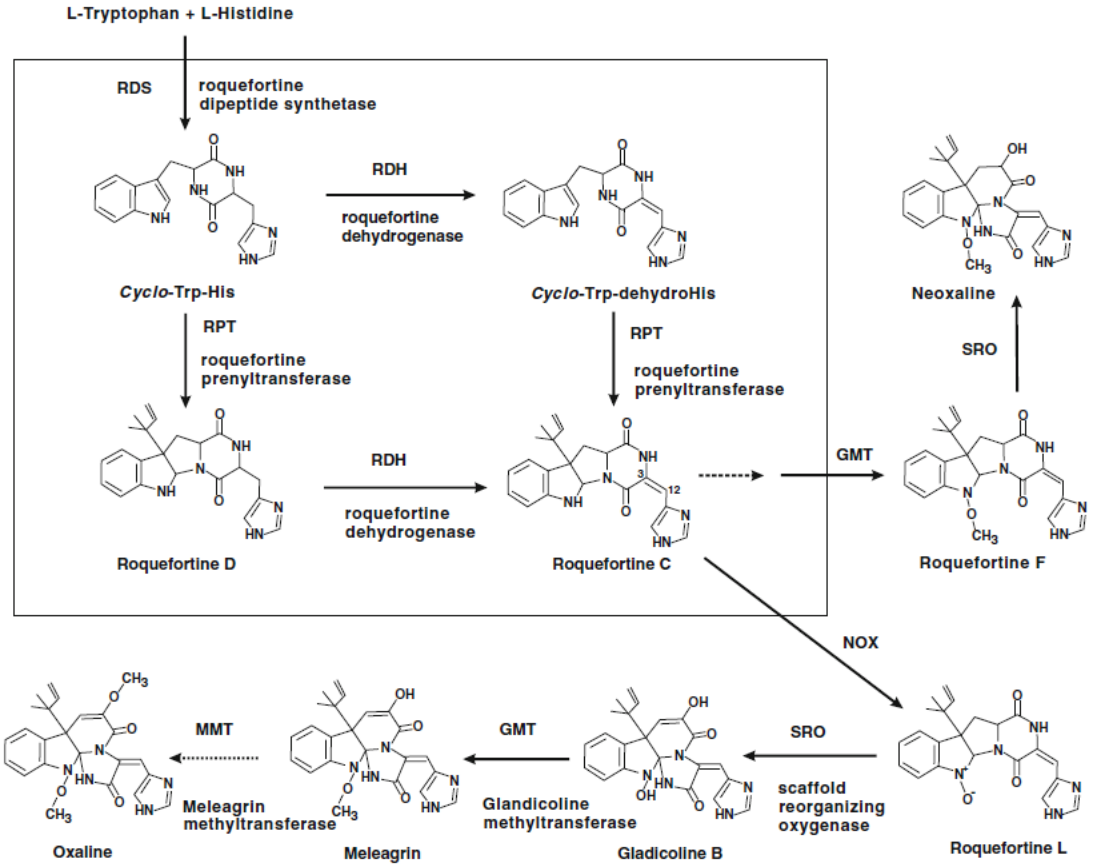


Figure 8 Biosynthetic pathway for synthesis of roquefortine C and meleagrin

Reproduced from Martín and Liras (2016).

Meleagrin has been shown to exhibit cytotoxicity against human HeLa cells (IC_{50} of 20 µg/mL) (Shang et al 2012). It has also been shown to be cytotoxic to a range of human cancer cell lines including the HepG2 liver cancer, Du145 prostate cancer and MDA-MB-231 epithelial breast cancer cell lines with IC_{50} values of 12, 5 and 11 µg/mL respectively (Shang et al 2012), and the A-549 lung and HL-60 leukaemia cell lines, with IC_{50} values of 20 and 7 µM, respectively (Du et al 2010). A recent study also showed that it has potent cytotoxicity against the human KB-3-1 cervix carcinoma cell line with an IC_{50} of 3.1 µM (Hamed et al 2021). It has also been found to inhibit the growth, migration and invasiveness of human breast cancer cell lines, and displays IC_{50} values of 1.9 – 8.9 µM against the MCF-7, MCF-7-dox, BT-474, SKBR-3, MDA-MB-468 and MDA-MB-231 breast cancer cell lines (Mady et al 2016), although Shang et al (2012) found no cytotoxicity to MCF-7 cells. Meleagrin also displays potent activity against the bacterium *Staphylococcus aureus* (minimum inhibitory concentration (MIC) of 0.25 mg/mL) (Hamed et al 2021).

No assessments specifically addressing inhalation exposure to meleagrin were identified during preparation of this report. However, as noted above it has been shown to be cytotoxic to the human A-549 lung cell line (Du et al 2010). Oral administration of meleagrin in mice has also been found to provide protection against bleomycin-induced pulmonary fibrosis (Elhady et al 2022).

4.5.5 Physcion

Physcion is an anthraquinone produced by both plants and fungi, including *Aspergillus fumigatus* and *Microsporum* species (Masi & Evidente 2020, XunLi et al 2019). It has been shown to be toxic to rat hepatocytes (Kang et al 2017, Panigrahi et al 2015), human liver cells (Molee et al 2018, Panigrahi et al 2015, Yu et al 2011), and baby hamster kidney cells *in vitro* (Chang et al 2014).

No studies specifically assessing inhalation exposure to physcion were identified during preparation of this report.

4.5.6 Penicillic acid

Penicillic acid was first isolated in 1913 from *Penicillium puberulum* and shown to be toxic to various laboratory animals (Ciegler et al 1971). It is known to be produced by a range of fungi including *Penicillium* and *Aspergillus* species (Frisvad 2018). It has been shown to display antimicrobial activity but is noted to be "too toxic for use in therapy", with LD_{50} values in mice of 100 mg/kg for subcutaneous administration (Ciegler et al 1971), and 250 and 530 mg/kg for intravenous and oral administration (Ueno & Ueno 1978). Penicillic acid has also been reported to be carcinogenic to rats and mice (Dickens & Jones 1961, Dickens & Jones 1965). In 1987 it was classified as a group III compound by the International Agency for Research on Cancer (IARC), which indicates it is not classifiable as to its carcinogenicity to humans[8]. This category is often used when "the evidence of carcinogenicity is inadequate in humans and inadequate or limited in experimental animals" but is also used for compounds that do not fall into any of the other categories[9]. The IARC did note that subcutaneous injection in rats and mice produced local sarcomas, but there were no case reports or epidemiological studies available for their assessment[10]. A 1977 study of penicillic acid toxicity in dogs found that intraperitoneal administration resulted in "hemorrhaging of the serosal surfaces of the abdomen" and signs of liver toxicity (Hayes et al 1977).

Limited information on the risk posed by inhalation was identified during preparation of this report. However, a study from 1986 found that penicillic acid is toxic to rat alveolar macrophages *in vitro*, and noted that this may "suggest the possibility of a respiratory hazard to agricultural workers exposed to contaminated grain" (Sorenson & Simpson 1986). Indeed, grain dusts from Belgian farms and storage companies were shown to contain up to 17 µg/g penicillic acid (Tangni & Pussemier 2007).

[8] https://monographs.iarc.who.int/list-of-classifications Accessed 31 August 2022
[9] https://inchem.org/documents/iarc/monoeval/eval.html Accessed 7 September 2022
[10] https://inchem.org/documents/iarc/vol10/penicillicacid.html Accessed 7 September 2022

4.5.7 Chaetoglobosin A

Chaetoglobosin A is a cytochalasin most notably produced by the biocontrol fungus *Chaetomium globosum* and exhibits potent antiparasitic, antimycotic and antitumor properties (Zhao et al 2022). It has been shown to be cytotoxic to human HeLa cells (Sekita et al 1982), and toxic to chick embryos (Veselý et al 1995), and human A549 lung and HepG2 liver cancer cell lines (Zhang et al 2021). Subcutaneous injection of chaetoglobosin A in rats and mice was found to result in high toxicity and rapid death (LD_{50} values of 6.5 and 18 mg/kg for male and female mice respectively), whereas oral administration of a single dose (50 – 400 mg/kg) had little effect (Ohtsubo et al 1978). Chaetoglobosin A has also been shown to display sublethal toxicity to boar spermatozoa and is noted to be an inhibitor of glucose transport (Salo et al 2020).

No studies specifically assessing toxicity of inhaled chaetoglobosin A were identified during preparation of this report, although as noted above it has been shown to be cytotoxic to human A549 lung cells (Zhang et al 2021).

4.5.8 Griseofulvin

Griseofulvin was first isolated from *Penicillium griseofulvum* in 1938 and is classified as a natural antibiotic (reviewed by Knasmüller et al (1997)). In 1997, it was reported to have been used for over 30 years as a pharmaceutical treatment for human dermatomycoses (Knasmüller et al 1997). However, it was also noted that "animal studies give clear evidence that it causes a variety of acute and chronic toxic effects, including liver and thyroid cancer in rodents, abnormal germ cell maturation, teratogenicity and embryotoxicity in various species" and "no sufficient data from human studies are available at present to exclude a risk to humans" (Knasmüller et al 1997). A report from 1978 noted that griseofulvin has an LD_{50} of 400 mg/kg for intravenous administration into rats (Ueno & Ueno 1978). Griseofulvin was reportedly withdrawn from the New Zealand market (and other countries) in 2002, "as it has been superseded by more effective and safer antifungal drugs"[11]. In 2001, griseofulvin was classified by the IARC as a group 2B compound, which indicates that it is possibly carcinogenic to humans[12].

No studies specifically assessing toxicity of inhaled griseofulvin were identified during preparation of this report.

4.5.9 3-Nitropropionic acid

3-Nitropropionic acid, also known as β-nitropropionic acid, is a neurotoxin produced by a range of plant, insect and fungal species, including *Aspergillus, Penicillium* and *Arthrinium* species (Burdock et al 2001), and is known to irreversibly inhibit the mitochondrial enzyme succinate dehydrogenase (Skogvold et al 2022, Urbanska et al 1998). Interestingly, it is naturally found in relatively high levels in karaka nuts, a traditional Māori food known to require substantial preparation in order to remove toxins (MacAskill et al 2015).

[11] https://dermnetnz.org/topics/griseofulvin Accessed 31 August 2022
[12] https://monographs.iarc.who.int/list-of-classifications Accessed 31 August 2022

3-Nitropropionic acid has been shown to be neurotoxic in mice and induces convulsions in a dose-dependent manner (Urbanska et al 1998). The LD_{50} in mice and rats is between 60 – 120 mg/kg (Burdock et al 2001). In possums, 3-nitropropionic acid displays an oral LD_{50} of 110 – 170 mg/kg, causing death in around 9 – 10 hours (Gregory et al 2000). Chronic treatment of baboons with 3-nitropropionic acid over 3 – 6 weeks was found to result in cognitive impairment and motor deficits similar to those seen in patients with Huntington's disease (Palfi et al 1996). Mild human exposure to 3-nitropropionic acid may result in a stomach-ache and vomiting, whereas severe exposure can result in dystonia (involuntary muscle contractions), coma and even death, and there is no known antidote (reviewed in Skogvold et al (2022)). 3-Nitropropionic acid has also been proposed to be responsible for fatal poisoning outbreaks in children in China caused by mouldy sugarcane (Liu et al 1992).

No studies specifically addressing toxicity of inhaled 3-nitropropionic acid were identified during preparation of this report.

4.5.10 Equisetin

This mycotoxin was first isolated in 1974 from the white mould *Fusarium equiseti*, but together with its epimer epiequisetin, has also been isolated from other *Fusarium* species including *F. pallidoroseum* and *F. heterosporum* (reviewed by Eze et al (2019)). Equisetin has been reported to have "an impressive biological activity profile including antibiotic and HIV inhibitory activity, cytotoxicity and mammalian DNA binding" (Burke et al 2005). It has also been shown to be a powerful inhibitor of rat mitochondrial ATPase activity (König et al 1993)

An early study noted that equisetin "is suspected to be a promoter of chronic environmental diseases including certain leukemias" (Turos et al 1989). Consistent with this, "*Fusarium equiseti* has repeatedly been identified in environments where several genetically unrelated individuals each developed leukemia" (Phillips et al 1989).

No studies specifically addressing toxicity of inhaled equisetin were identified during preparation of this report.

4.5.11 Chanoclavine

Chanoclavine is an early intermediate in the ergot alkaloid biosynthetic pathway and has been shown to accumulate to varying levels in ergot alkaloid-producing species (Coyle & Panaccione 2005). Chanoclavine has been shown to be present in small amounts in the conidia (spores) of *Aspergillus fumigatus* (Coyle & Panaccione 2005), a species often found in indoor environments (Araujo et al 2010, Hedayati et al 2010).

The potential toxicity of chanoclavine to mammals was assessed in an acute oral toxicity study in mice (Finch et al 2019). This showed that the median lethal dose was greater than 2000 mg/kg, which allows it to be classified as a category 6.1E chemical, the lowest hazard class under the New Zealand Hazardous Substances and New Organisms classification system (Finch et al 2019). However, some of the mice did initially display signs of neurotoxicity, including tremors and "splaying of the back legs", but this resolved within 24 hours (Finch et al 2019). The authors also conducted a three-week feeding study which

found an initial reduction in food consumption that resolved within one week, but “no toxicologically significant effects on gross pathology, histology, hematology, or blood chemistry” (Finch et al 2019). As such, the authors reported that chanoclavine has low toxicity. However, in a recent review it was noted that “further research is necessary, due to the reported toxicity of ergot alkaloids to mammals, including human” (Adamski et al 2020).

No additional studies of the toxicity of chanoclavine, including potential toxicity of inhaled chanoclavine, were identified during preparation of this report.

5. ASSESSING MYCOTOXIN EXPOSURE

Human mycotoxin exposure is usually assessed using biomonitoring studies, dietary surveys and/or analysis of foodstuffs (Berzina et al 2022, Gracia-Lor et al 2020). However, these methods are generally time-consuming and expensive (Berzina et al 2022, Gracia-Lor et al 2020). Recently, wastewater-based epidemiology has been developed as a complementary tool for estimating population-level mycotoxin exposure (Berzina et al 2022, Gracia-Lor et al 2020). This section will provide an overview of the biomonitoring and WBE approaches used to estimate mycotoxin exposure and highlight those studies which have assessed exposure to the mycotoxins most frequently identified in studies of the indoor environment cited in this report.

5.1 BIOMONITORING

Urinary biomonitoring has already been employed in New Zealand to assess exposure to a range of heavy metals, phthalate metabolites, cotinine, phenols and fluoride ('t Mannetje et al 2018). However, no biomonitoring of mycotoxin exposure has been conducted in New Zealand.

Urinary biomonitoring has been employed around the globe to assess human exposure to a range of mycotoxins, as summarised in Table 5, and detailed in full in Appendix Table 19. Biomonitoring may allow for estimation of not only dietary exposure but also environmental exposure through inhalation or direct skin contact (Föllmann et al 2016, Habschied et al 2021). Precedent exists to screen urine for inhalation exposure to mycotoxins as it has been used to screen for occupational exposure in grain mill workers in Germany (Föllmann et al 2016), feed handling workers in Brazil (Franco & Oliveira 2022), workers in a grain elevator in France (Ndaw et al 2021) and feed mill workers in Italy (Ferri et al 2017). Additionally, several companies market urine spot tests to screen for mycotoxin exposure, with inhalation noted as a potential exposure route, including NutriPATH in Australia[13], RealTime Labs in the US[14] and FxMed in New Zealand and Australia[15].

Seventy-nine urinary biomonitoring studies from around the world were examined during preparation of this report. The number of participants in these studies ranged from 10 to 2,212. Mycotoxins considered in these studies were primarily those encountered through dietary exposure and included aflatoxins, fumonisins, ochratoxins, trichothecenes, zearalenones, citrinin, enniatins, *Alternaria* toxins and ergot alkaloids.

[13] https://www.nutripath.com.au/product/mycotoxin-exposure-urine-test-code-3413-3414/ Accessed 6 September 2022
[14] https://realtimelab.com/mycotoxin-testing/ Accessed 6 September 2022
[15] https://fxmed.co.nz/product/functional-testing/the-great-plains-lab/gpl-mycotox/ Accessed 6 September 2022

Table 5 Summary of the main classes of mycotoxins assessed by urinary biomonitoring studies

Mycotoxin class	Number of studies assessing exposure
Ochratoxins	42
Trichothecenes	41
Aflatoxins	33
Zearalenones	28
Fumonisins	23
Citrinin	13
Alternaria toxins	8
Enniatins	8
Others	6
Ergot alkaloids	1

Based on those studies identified during preparation of this report.

With regards to those mycotoxins most frequently identified in studies of the indoor environment cited in this report, alternariol, alternariol monomethyl ether, beauvericin, enniatins A, A1, B and B1, roquefortine C, sterigmatocystin and verrucarol were all found to have been assessed in at least one urinary biomonitoring study, as summarised in Table 6. Although most of these studies only directly measured levels of the parent mycotoxin, five studies pre-treated the urine samples with β-glucuronidase or a β-glucuronidase/aryl sulfatase mixture in order to reconvert conjugated mycotoxins back to the parent form (refer Table 6) (Šarkanj et al 2018). This was primarily done as there is limited availability of mycotoxin glucuronide conjugates for use as analytical standards (Šarkanj et al 2018). Additionally, the studies of Rodríguez-Carrasco et al (2018) and (2020) assessed both enniatin B/B1 and their phase I metabolites.

Alternariol, alternariol monomethyl ether, enniatins A1, B and B1, roquefortine C, sterigmatocystin and verrucarol have all been detected in urine samples taken during these various biomonitoring studies, although enniatin A1 was not detected in quantifiable amounts. The incidence of detection varied quite considerably between the different studies, potentially due at least in part to some employing enzymatic pre-treatment. The incidence of detection of enniatin B ranged from 2 – 84%, enniatin B1 ranged from 43 – 94% and alternariol ranged from 7 – 52%. Alternariol monomethyl ether was detected with the highest incidence, with two Chinese studies (n = 2212 and n = 135) reporting incidence of detection of 96% at concentrations ranging from below the limit of quantification to 2.0 and 0.15 ng/mL respectively (Qiao et al 2022, Qiao et al 2020). However, in the EFCOVAL study which includes samples from Belgium, the Czech Republic, France, the Netherlands and Norway, the incidence was only 7%. It has been suggested that the difference in incidence could be due to “differences in nutritional habits and quality of consumed foodstuffs” (Rodríguez-Carrasco et al 2018). Roquefortine C, enniatin A1, sterigmatocystin and verrucarol were all detected at incidences lower than 20%, whilst beauvericin and enniatin A were not detected in any of the tested samples.

Table 6 Results of biomonitoring for mycotoxins most frequently identified in the indoor environment

Mycotoxin	No. studies assessing/No. detected	Country	Participants	Enzymatic pre-treatment	Incidence	Concentration (ng/mL)	Concentration (µg/g creatinine)	Reference
Alternariol	8/6	China	2212	β-gluc/arylS	11%	< LOQ – 32	ND	Qiao et al (2022)
		EFCOVAL	600	-	7%	0.025 – 1.6	ND	De Ruyck et al (2020)
		China	269	β-gluc/arylS	38%	0.06 – 46	0.04 - 44	Fan et al (2021)
		China	135	β-gluc/arylS	52%	0.06 – 2.6	0 – 5.0	Qiao et al (2020)
		Nigeria	120	β-gluc	7%	0.03 – 0.20	ND	Šarkanj et al (2018)
		Portugal	94	-	29%	< LOQ - 25	0 - 17	Martins et al (2019)
Enniatin B	6/4	Italy	300	-	84%	0.006 – 0.39	ND	Rodríguez-Carrasco et al (2018)
		Haiti Germany Bangladesh	142 50 95	-	3% 14% 2%	0.021 – 0.065 0.010 – 0.014 0.012 – 0.019	0.029 (mean) 0.017 (mean) 0.015 (mean)	Gerding et al (2015)
		Germany	101	-	20%	< LOQ	ND	Gerding et al (2014)
		Spain	10	-	80%	< LOQ – 0.54	ND	Escrivá et al (2017)
Alternariol monomethyl ether	7/4	China	2212	B-gluc/arylS	96%	< LOQ – 2.0	0 – 1.8	Qiao et al (2022)
		EFCOVAL	600	-	7%	0.13 – 22	ND	De Ruyck et al (2020)
		China	269	β-gluc/arylS	49%	0.02 – 0.8	0.01 – 2.1	Fan et al (2021)

		China	135	β-gluc/arylS	96%	< LOQ – 0.15	0.001 – 0.083	Qiao et al (2020)
Enniatin B1	5/3	EFCOVAL	600	-	43%	0.30 – 18	ND	De Ruyck et al (2020)
		Italy	300	-	94%	0.007 – 0.43	ND	Rodríguez-Carrasco et al (2020)
		Spain	10	-	60%	< LOQ – 0.34	ND	Escrivá et al (2017)
Enniatin A	3/0							
Enniatin A1	3/1	Spain	10	-	10%	< LOQ	ND	Escrivá et al (2017)
Beauvericin	3/0							
Sterigmatocystin	3/2	EFCOVAL	600	-	16%	0.0047 – 8.3	ND	De Ruyck et al (2020)
		Qatar	559	β-gluc	1%	< LOQ		Al-Jaal et al (2021)
Roquefortine C	3/2	EFCOVAL	600	-	7%	0.0021 – 40	ND	De Ruyck et al (2020)
		Qatar	559	β-gluc	4%	< LOQ – 0.33	ND	Al-Jaal et al (2021)
Verrucarol	1/1	EFCOVAL	600	-	2%	0.0016 – 0.0047	ND	De Ruyck et al (2020)

EFCOVAL, European Food Consumption Validation project – includes samples from Belgium, the Czech Republic, France, the Netherlands and Norway. LOQ, limit of quantification; ND, not determined.

5.2 WASTEWATER-BASED EPIDEMIOLOGY

Wastewater-based epidemiology is an emerging technique in which population-level exposure to a particular chemical or microbe is assessed through measurement of indicators, or biomarkers, of exposure present in municipal wastewater. It is based on the premise that many of the chemicals and microbes we are exposed to in our environment can be absorbed and ultimately excreted either unchanged or in a metabolised form in urine and/or faeces (reviewed by Eaton et al (2021)).

A WBE approach has already been used to assess population-level exposure to selected mycotoxins in Italy, Spain (Gracia-Lor et al 2020), and Latvia (Berzina et al 2022). In the Gracia-Lor et al (2020) study LC-MS/MS was used to screen untreated wastewater from two cities in Italy and Spain for eleven different compounds chosen based on a review of information from urinary biomonitoring studies and occurrence in urban wastewater. The populations serviced by the four wastewater treatment plants investigated ranged from 95,000 to 1,080,000. To be selected, the biomarker had to be present in urine in µg/L or mg/L concentrations, have a frequency of detection in reported biomonitoring studies of ≥ 40%, have already been reported as present in urban wastewater and have available analytical standards to allow for quantification (Gracia-Lor et al 2020). The final biomarkers chosen are shown in Figure 9. None of these correspond with the mycotoxins most frequently reported in studies of the indoor environment cited in this report and are likely to relate predominantly to dietary exposure.

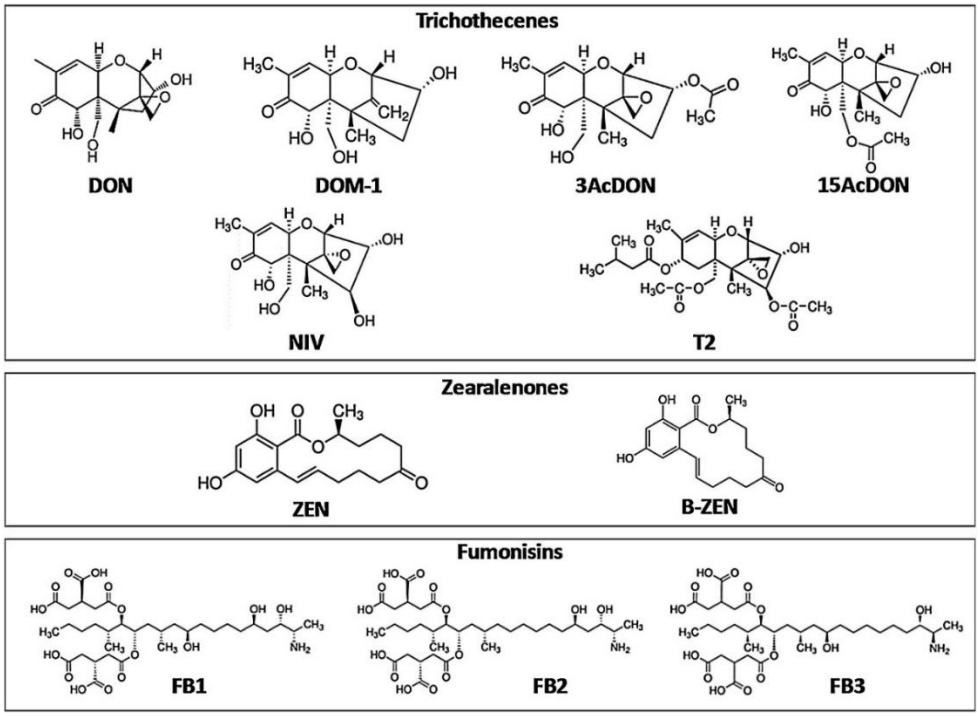


Figure 9 Mycotoxin targets of the Gracia-Lor et al (2020) study

Reproduced from Gracia-Lor et al (2020). DON, deoxynivalenol; NIV, nivalenol; DOM-1, deepoxy-deoxynivalenol; T-2, T-2-toxin; 3AcDON, 3-acetyl-deoxynivalenol; 15AcDON, 15-acetyl-deoxynivalenol; ZEN, zearalenone; β–ZEN, β-zearalenol; FB1-3, fumonisins B1-B3.

The only biomarkers detected in this study were deoxynivalenol and the three fumonisins (Table 7). All four of these biomarkers were detected in the Italian samples, but only deoxynivalenol and fumonisin B3 were detected in samples taken in Spain (Gracia-Lor et al 2020).

Table 7 Detection of mycotoxins in urban wastewater in Italy and Spain

	Italy		Spain	
	Milan	Mozzanica	Castellon	Central Spain
Pop.	1,080,000	95,000	180,000	383,235
	(n = 7)	(n = 8)	(n = 7)	(n = 7)
DON	34.6 ± 4.2 (100%)	32.8 ± 11.2 (100%)	46.6 ± 16.3 (100%)	32.2 ± 9.3 (100%)
FB1	5.9 ± 0.7 (100%)	104.1 ± 52.6 (100%)	< LOQ (0%)	< LOQ (0%)
FB2	1.4 ± 0.1 (100%)	7.8 ± 3.0 (100%)	< LOQ (0%)	< LOQ (0%)
FB3	< LOQ (100%)	5.5 ± 2.6 (100%)	2.3 ± 1.7 (100%)	< LOQ (0%)

Adapted from Gracia-Lor et al (2020). Concentrations in ng/L (mean ± standard deviation), frequency of detection indicated in brackets. Pop, population serviced by the wastewater treatment plant.

The authors note that their estimates of deoxynivalenol exposure were similar to those determined by biomonitoring studies and foodstuff analyses, validating the use of WBE as a complementary method (Gracia-Lor et al 2020). The authors also highlighted several areas of uncertainty in the use of WBE for mycotoxins that need to be considered if it were to be adopted as a method for assessing mycotoxin exposure in New Zealand. These areas of uncertainty are:

1. Information on human metabolism of mycotoxins is required to back-calculate exposure. However, there are limited pharmacological studies on the urinary excretion of mycotoxins making it difficult to determine back-calculation factors.
2. Where the parent mycotoxin is used as a biomarker rather than a human-specific metabolite the potential for other, non-human sources must be considered.
3. Information is lacking on the stability of mycotoxins in-sewer and further investigation into the potential biotransformation of these compounds under different conditions in-sewer is required.
4. There is a need for research into the potential “hydrolysis of glucuronides or other conjugated metabolites to the free form of the parent compound in raw wastewater or during wastewater treatment” (Gracia-Lor et al 2020).
5. Their study only assessed the potential of WBE for monitoring mycotoxin exposure on a small scale and larger scale studies are required to refine the methodology and determine if it is a suitable complementary method to biomonitoring (Gracia-Lor et al 2020).

More recently, WBE has been used to assess exposure to mycotoxins in Riga, Latvia (Berzina et al 2022). In this study, the authors specifically assessed the presence of deoxynivanelol, enniatins A, A1, B, B1 and beauvericin in wastewater arriving at the inlet of the Riga wastewater treatment plant which services a population of 697,000 people (Berzina et al 2022). As part of this study, they assessed the stability of these mycotoxins in wastewater after two and three weeks at 4°C and -18°C, as shown in Table 8.

Table 8 Stability of mycotoxins in wastewater

Compound	Difference after 2 Weeks at 4°C (%)	Difference after 3 Weeks at 4°C (%)	Difference after 2 Weeks at -18°C (%)	Difference after 3 Weeks at -18°C (%)
DON	-2,2	Not Tested	-13	Not tested
BEA	-35	-35	-47	-41
ENNA	-41	-31	-25	-11
ENNA1	-9	+24	-71*	-19
ENNB	+3	+10	-15	+4
ENNB1	+5	0	-15	+24

Reproduced from Berzina et al (2022). Stability calculated as a percentage of the concentrations measured at t_0, immediately after spiking with 50 ng/L deoxynivalenol, 50 ng/L beauvericin or 5 ng/L of the different enniatins. *Assumed as random error. DON, deoxynivalenol; BEA, beauvericin; ENNA, enniatin A; ENNA1, enniatin A1; ENNB, enniatin B; ENNB1, enniatin B1.

To assess mycotoxin exposure, 29 samples were collected over a six-week period. All samples contained deoxynivalenol and enniatins B and B1, whilst 90% and 86% contained enniatins A and A1 respectively (Table 9). Beauvericin was only detected in four samples (14%), and all detections were at concentrations below the limit of quantification (Berzina et al 2022)

Table 9 Detection of mycotoxins in urban wastewater in Riga, Latvia

	Frequency of Detection (>LOD)	Average, ng/L	Range, ng/L	Median, ng/L	LOD, ng/L	LOQ, ng/L	Precision, %
DON	100%	51.7	23.2–76.9	52.9	1.9	6.4	6
ENNA	90%	3.0 *	LOQ–16.7	2.0 *	0.12	0.40	3
ENNA1	86%	4.2 *	LOQ–27.7	2.5 *	0.14	0.47	4
ENNB	100%	4.6	0.6–9.9	4.1	0.04	0.15	1.3
ENNB1	100%	3.9	1.4–10.1	3.4	0.13	0.43	3
BEA	14%	<LOQ	<LOQ	<LOQ	0.04	0.13	7

* The mean values were calculated using LOQ/2 when the measured values were below LOQ.

Reproduced from Berzina et al (2022). DON, deoxynivalenol; ENNA, enniatin A; ENNA1, enniatin A1; ENNB, enniatin B; ENNB1, enniatin B1; BEA, beauvericin; LOD, limit of detection; LOQ, limit of quantification.

The study also quantified the presence of 5-hydroxyindoleacetic acid (5-HIAA) in the wastewater samples and used this as a population biomarker, allowing them to estimate intake of deoxynivalenol by the inhabitants of Riga. This biomarker is the main serotonin metabolite and has been recommended as a suitable population biomarker with considerable information known about its excretion (Berzina et al 2022).

With regards to the enniatins and beauvericin, Berzina et al (2022) note that there is very little information on the "kinetics of absorption and excretion of enniatins and beauvericin". Given there was no information on the urinary excretion of enniatins on which daily intake calculations could be based, the authors assumed it to be in the range of 5 – 50% and calculated provisional daily intakes based on 5, 25 and 50% assumed excretion factors, as shown in Table 10.

Table 10 Provisional daily intake of enniatins

	ENNA			ENNA1		
Assumed Excretion Factors	5%	25%	50%	5%	25%	50%
Average	0.25	0.050	0.025	0.36	0.072	0.036
Max	1.18	0.24	0.12	1.95	0.39	0.19
Min	0.012	0.0024	0.0013	0.017	0.0034	0.0017
Median	0.15	0.029	0.015	0.21	0.042	0.021
	ENNB			**ENNB1**		
Average	0.39	0.079	0.039	0.35	0.069	0.035
Max	0.84	0.17	0.084	1.05	0.21	0.105
Min	0.048	0.0097	0.0049	0.109	0.022	0.0109
Median	0.34	0.067	0.034	0.30	0.060	0.030

Values all in µg/kg bw day.

Results of this study demonstrate that enniatins can be detected and quantified using WBE. However, additional research on the excretion of enniatins is needed to determine an accurate excretion factor. Additional research on the metabolism and excretion of beauvericin is required to determine whether this truly represents low exposure to beauvericin in the assessed populations, or whether it is highly metabolised and there is a more suitable metabolite which could be used to assess exposure.

6. WASTEWATER-BASED EPIDEMIOLOGY TO ASSESS INDOOR MYCOTOXIN EXPOSURE

In this section, the feasibility of measuring indoor mycotoxin exposure using WBE will be assessed. Based on prevalence in studies of mycotoxins present in the indoor environment, twenty-three mycotoxins were chosen as potential candidates due to their detection in five or more studies (refer Table 4). However, chanoclavine was excluded from further assessment due to lack of evidence suggesting potential toxicity to humans. The remaining twenty-two candidates were assessed based on what is known about their metabolism, excretion in urine and/or faeces, presence in wastewater and presence in food. Their overall suitability for WBE is discussed.

6.1 STERIGMATOCYSTIN

Sterigmatocystin was the most frequently reported mycotoxin in the studies of the indoor environment assessed during preparation of this report, detected in 19 different studies spanning at least 12 different countries including New Zealand. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, floor and settled dust, and indoor air. Three biomonitoring studies assessing sterigmatocystin in urine were identified. However, it was only detected in two of these studies and with low incidence of detection (1 – 16%; Table 6). The detected concentrations were also low, being below the limit of quantification in one study (Al-Jaal et al 2021) and 0.0047 – 8.3 ng/mL in the other study (De Ruyck et al 2020).

A study in rats assessed the excretion of sterigmatocystin and its equivalents in urine and faeces using ^{14}C-sterigmatocystin (Walkow et al 1985). Up to 10% of the orally administered dose was found to be excreted in urine, with at least 90% of this excreted within the first 48 hours. Fecal excretion was significantly different between male and female rats, with males excreting an average of 90% of the oral dose in their faeces and females an average of only 65% of the dose (Walkow et al 1985). This preferential excretion of sterigmatocystin equivalents in faeces rather than urine may explain why only low levels were detected in the human urinary biomonitoring studies (Al-Jaal et al 2021, De Ruyck et al 2020). Although, the low level of detection may simply be due to low exposure of the tested individuals to sterigmatocystin. A separate ELISA-based study in rats found that excretion of sterigmatocystin metabolites in urine was rapid, with 86% of the total urinary excretion within 24 hours (Olson & Chu 1993). As such, the authors noted that “monitoring the urine metabolites of exposed individuals would require urine to be collected soon after exposure” (Olson & Chu 1993). Urinary excretion was also found to be dose-dependent in rats (Olson & Chu 1993).

Examination of urinary excretion of sterigmatocystin by vervet monkeys found that more than 50% of the sterigmatocystin excreted in urine is conjugated to glucuronic acid, and the authors note that it is “without doubt, that unaltered sterigmatocystin is conjugated with

glucuronic acid before excretion in the urine" (Thiel & Steyn 1973). Similarly, a study in cattle noted that sterigmatocystin is "extensively conjugated in the liver, presumably to glucuronic acid, as only trace amounts of the free mycotoxin could be detected in urine", whilst pre-incubation with β-glucuronidase/ arylsulfatase resulted in measurable sterigmatocystin concentrations of up to 140 ng/g creatinine (Fushimi et al 2014). Interestingly, in the biomonitoring study of Al-Jaal et al (2021) the urine samples were treated with β-glucuronidase but sterigmatocystin concentrations were still below the limit of quantification.

Whilst evidence from animal studies suggest WBE for sterigmatocystin may be feasible given it is excreted in faeces and urine, the observation that the majority of sterigmatocystin is excreted as a glucuronide conjugate may pose a problem as "in general, glucuronide compounds are very quickly transformed in wastewater and hence are not suitable to be used as biomarkers in WBE" (Banks et al 2018). However, these compounds are transformed back to the parent sterigmatocystin (eg, deconjugated) then it could still be measured using unmetabolised sterigmatocystin as the biomarker.

Regardless of whether the glucuronide conjugates are transformed to the parent sterigmatocystin, it may still be possible to screen for unmetabolised sterigmatocystin, as De Ruyck et al (2020) were able to detect sterigmatocystin in urine samples without glucuronidase pre-treatment, albeit at low concentrations (0.0047 – 8.3 ng/mL). However, no studies reporting the presence of sterigmatocystin in wastewater were identified during preparation of this report. The stability of sterigmatocystin or its metabolites in wastewater would need to be assessed prior to commencement of WBE. If unmetabolised sterigmatocystin was measured, the contribution of non-human sources to its presence in wastewater would need to be considered. Sterigmatocystin has been reported in a variety of foodstuffs including various nuts and spices, cheese, cereals and cereal products, beer and green coffee beans and animal feed (Cressey & Pearson 2014a, JECFA 2017). Disposal of food waste contaminated with sterigmatocystin to the wastewater network (eg, via disposal of mouldy foods through kitchen sink waste disposal systems) could therefore add to levels measured using WBE. However, little is known about the occurrence of sterigmatocystin in food or crops in New Zealand (Cressey & Pearson 2014a). Although it was noted that the main sterigmatocystin-producing species, *A. versicolor* and *A. nidulans* have not been reported in New Zealand (Cressey & Pearson 2014a). Contribution of sterigmatocystin from cleaning of contaminated surfaces/materials should also be considered if unmetabolised sterigmatocystin were chosen as a biomarker for WBE. A reference standard is commercially available for sterigmatocystin to allow for quantification if it were to be measured (CAS: 10048-13-2)[16].

As there are known dietary sources of sterigmatocystin, to distinguish dietary and indoor exposure, it would be useful to establish a baseline level of dietary exposure using biomonitoring or similar. This could then be subtracted from measured levels to allow for estimation of indoor exposure. However, establishing dietary exposure levels will be complex as different subpopulations may vary in their dietary preferences, which can influence mycotoxin exposure (Bennett & Klich 2003) and there may be seasonal differences in dietary mycotoxin exposure. As yet, no biomonitoring for sterigmatocystin has been conducted in New Zealand and the 2014 Mycotoxin Surveillance Program report did not identify any estimates of dietary exposure to sterigmatocystin (Cressey & Pearson 2014a). In their recent

[16] https://www.sigmaaldrich.com/NZ/en/product/sial/32609 Accessed 8 September 2022

assessment of sterigmatocystin, EFSA concluded that there was insufficient occurrence data available to perform a dietary exposure assessment (EFSA 2013).

Overall, it may be possible to use WBE to monitor for indoor exposure to sterigmatocystin. However, preliminary work is required to confirm if unmetabolised sterigmatocystin can be reliably detected in wastewater, or whether a more suitable biomarker is available. Targeting of WBE sampling to small populations where high indoor exposure is suspected (eg, flood-affected communities) may result in higher detection due to less dilution from unexposed individuals. Establishment of baseline dietary exposure levels for sterigmatocystin would also be useful to allow for specific estimation of environmental exposure.

6.2 ENNIATINS AND BEAUVERICIN

Berzina et al (2022) noted that “very little information is currently available about the kinetics of absorption and excretion of enniatins and beauvericin in any kind of species” (Berzina et al 2022). As such, in their WBE study they used assumed excretion factors of 5, 25 and 50% to calculate provisional daily intakes, as discussed above. A 2014 report by EFSA noted that beauvericin and enniatins are “rapidly metabolised to a range of uncharacterised metabolites” (EFSA 2014).

Enniatins and beauvericin are known to have high co-occurrence in food, which is not unexpected given they are produced by the same metabolic pathway (EFSA 2014). These toxins were also found to co-occur in several of the indoor environment studies assessed during preparation of this report (Lindemann et al 2022, Peitzsch et al 2012, Täubel et al 2011, Vishwanath et al 2009, Vishwanath et al 2011), including the New Zealand BRANZ study (Plagmann et al 2021). As such, it may be sufficient to just assess exposure to one of these toxins using WBE.

6.2.1 Enniatin A

Enniatin A was detected in nine different studies of the indoor environment assessed during preparation of this report, spanning at least six different countries including New Zealand. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15). Three biomonitoring studies assessing enniatin A in urine were identified. However, it was not detected in any of these studies (Escrivá et al 2017, Kyei et al 2022, Warensjö Lemming et al 2020). This may be due to preferential excretion of unmetabolised enniatin A in faeces rather than urine, as a study in rats showed that after administration of a mixture of enniatins containing A, A1, B and B1, the enniatin A concentration in urine was below the limit of quantification, whereas it was detected in faeces with an average concentration of 11 ng per gram of faeces (Escrivá et al 2015). However, another study in rats failed to detect enniatin A in urine or faeces during a four-week feeding study (Juan et al 2014). This non-detection may also be due to extensive metabolism of enniatin A as noted above (EFSA 2014). All three biomonitoring studies only assessed the presence of the parent enniatin A and none employed enzymatic pre-treatment to convert metabolites back to the parent form (Escrivá et al 2017, Kyei et al 2022, Warensjö Lemming et al 2020).

A 2021 review of enniatins and livestock noted that further studies are needed to “check the presence of various phase I metabolites in excreta because demethylated, oxidated, hydroxylated and carbonylated metabolites were recently tentatively identified in human urine samples” (Křížová et al 2021). This is referring to two studies by Rodríguez-Carrasco et al. which identified phase I metabolites for enniatins B and B1 in human urine (Rodríguez-Carrasco et al 2018, Rodríguez-Carrasco et al 2020).

Enniatin A was assessed in the Berzina et al (2022) WBE study and detected with an incidence of 90% at concentrations ranging from below the limit of quantification to 17 ng/L. Assessment of the stability of enniatin A in wastewater by Berzina et al (2022) showed that its concentration reduced by approximately 30 – 40% over three weeks at 4°C and approximately 10 – 25% over three weeks at -18°C (Table 8).

Enniatin A has been reported in a range of foods including fresh and dry pasta, mixed cereals (EFSA 2014), breakfast and infant cereals, wheat, maize, barley, rice, oats, rye, sorghum and grain-based products (Santini et al 2012). As such, contaminated food waste discharged to the wastewater network could add to the level of enniatin A detected using WBE. Additionally, in contrast to the Berzina et al (2022) study which was aimed at primarily assessing dietary mycotoxin exposure, WBE to assess indoor exposure to enniatin A would require establishment of a baseline level of dietary exposure in New Zealand, which could then be subtracted off measured levels. However, as noted above this may be difficult due to preferential excretion of enniatin A in faeces rather than urine. It may be possible to conduct faecal rather than urinary biomonitoring, as has been done for exposure to hexachlorobenzene and its metabolites (To-Figueras et al 2000) and polybrominated diphenyl ether (PBDE) flame retardants (English et al 2017). Enniatins have not been addressed by the Mycotoxin Surveillance Program, so little is known about their presence in foods in New Zealand.

Similar studies to those conducted by Rodríguez-Carrasco et al. could be undertaken to determine if there are metabolites of enniatin A in human urine that could act as a biomarker for enniatin A exposure, eliminating the impact of non-human sources of enniatin A in wastewater on exposure assessment. If unmetabolised enniatin A were to be measured, a reference standard is commercially available, allowing quantification (CAS: 2503-13-1)[17].

Overall, enniatin A is a promising candidate for WBE. Unmetabolised enniatin A has been shown to be measurable using WBE, so could be screened for in New Zealand to assess exposure to enniatin A in the indoor environment. However, as for sterigmatocystin, it would be useful to establish baseline dietary exposure levels to allow for estimation of the contribution from indoor exposure. Although only low levels of enniatin A were detected by Berzina et al (2022), targeting of WBE to sub-populations where high indoor exposure is suspected (eg, areas with substantial water-damage or dampness issues) may result in higher detection levels. The possibility of screening for a metabolite of enniatin A is also an area worth exploring as this would eliminate the problem caused by non-human sources of enniatin A contributing to levels detected using WBE.

[17] https://www.sigmaaldrich.com/NZ/en/product/sigma/e9661 Accessed 8 September 2022

6.2.2 Enniatin A1

Enniatin A1 was detected in nine different studies of the indoor environment assessed during preparation of this report, spanning at least six different countries including New Zealand. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15). Three biomonitoring studies assessing enniatin A1 in urine were identified (Escrivá et al 2017, Kyei et al 2022, Warensjö Lemming et al 2020). However, it was only detected in one of these studies with an incidence of detection of 10% and concentrations below the limit of quantification (Escrivá et al 2017). Similar to enniatin A, this may be due to preferential excretion of unmetabolised enniatin A1 in faeces rather than urine as in the rat study detailed above, the enniatin A1 concentration in urine was below the limit of quantification whereas it was detected in faeces with an average concentration of 8.1 ng per gram of faeces (Escrivá et al 2015). However, this may also be due to extensive metabolism of enniatin A1 as noted above. As for enniatin A, the three biomonitoring studies only assessed the presence of the parent enniatin A1 and none employed any enzymatic pre-treatment to convert metabolites back to the parent form (Escrivá et al 2017, Kyei et al 2022, Warensjö Lemming et al 2020).

Enniatin A1 was assessed in the Berzina et al (2022) WBE study and detected with an incidence of 86% at concentrations ranging from below the limit of quantification to 28 ng/L. Results of assessment of the stability of enniatin A1 in wastewater by Berzina et al (2022) suggest that it is relatively stable at 4°C (for three weeks), and its levels reduced by around 20% after three weeks at -18°C (Table 8).

Enniatin A1 has been reported in the same range of foodstuffs as enniatin A (EFSA 2014, Santini et al 2012), and as such, potential contribution of contaminated food waste discharged to the wastewater network on levels of enniatin A1 detected using WBE must be considered. As discussed for enniatin A, specific assessment of indoor exposure using WBE would require preliminary work to establish a baseline level of dietary exposure in New Zealand. Ideally, a human metabolite of enniatin A1 should also be identified to eliminate the impact of non-human sources of enniatin A1 in wastewater on exposure assessment. If unmetabolised enniatin A1 were to be measured, a reference standard is commercially available (CAS: 4530-21-6)[18].

Overall, as for enniatin A, enniatin A1 is a promising candidate for WBE. Unmetabolised enniatin A1 has been shown to be measurable using WBE, so could be screened for in New Zealand to assess exposure in the indoor environment. However, this would first require estimation of baseline dietary exposure levels. Similar to enniatin A, targeting of WBE to sub-populations where high indoor exposure is suspected may increase detection levels. Identification of a suitable metabolite of enniatin A1 to use as a biomarker for WBE would also be preferable to eliminate the problem caused by non-human sources of enniatin A1 present in wastewater.

6.2.3 Enniatin B

Enniatin B was detected in 10 different studies of the indoor environment assessed during preparation of this report, spanning at least seven different countries including New Zealand.

[18] https://www.sigmaaldrich.com/NZ/en/product/sigma/e5161 Accessed 8 September 2022

Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15). Six biomonitoring studies assessing enniatin B in urine were identified. However, it was only detected in four of these studies and with highly variable incidence of detection ranging from 2 – 84%. The detected concentrations ranged from below the limit of quantification to 0.54 ng/mL (Escrivá et al 2017, Gerding et al 2015, Gerding et al 2014, Rodríguez-Carrasco et al 2018). Again, like enniatins A and A1, the low concentrations of enniatin B detected in these biomonitoring studies may be due to preferential excretion of unmetabolised enniatin B in faeces rather than urine. In the rat study detailed above, the enniatin B concentration in urine was below the limit of quantification whereas it was detected in faeces with an average concentration of 170 ng per gram of faeces (Escrivá et al 2015). A separate preliminary study orally administered a mixture of enniatins A, A1, B and B1 to rats and found that 5 – 10% of the enniatin B dose was excreted unmetabolised in urine within 24 hours (Koivisto et al 2015). In contrast, a study in mice found that enniatin B was undetectable in urine after intraperitoneal injection (Rodríguez-Carrasco et al 2016). Enniatin B has been shown to be extensively metabolised by human liver microsomes, producing 12 different phase I biotransformation products (Ivanova et al 2011). Enniatin B and its phase I metabolites have been identified in human urine (Rodríguez-Carrasco et al 2018) (Table 11). Unmetabolised enniatin B was identified in 84% of the samples, at concentrations ranging from 0.006 – 0.39 ng/mL (Rodríguez-Carrasco et al 2018).

Enniatin B was assessed in the Berzina et al (2022) WBE study and detected in all samples with concentrations ranging from 0.6 – 9.9 ng/L. Results of assessment of the stability of enniatin B in wastewater by Berzina et al (2022) suggest that it is relatively stable at 4°C and -18°C for up to three weeks (Table 8).

Enniatin B has been reported in a range of foodstuffs including wheat, barley, oats, rye, maize, breakfast and infant cereals, rice and cereal products (Santini et al 2012), and as such, potential contribution of contaminated food waste discharged to the wastewater network on levels of enniatin B detected using WBE must be considered. To overcome this problem, it may be possible to use one of the human phase I enniatin B metabolites identified by Rodríguez-Carrasco et al (2018) as a biomarker. However, this would require preliminary studies to assess its detection and stability in wastewater. Additionally, Rodríguez-Carrasco et al (2018) noted that there are no standards available for the enniatin B metabolites, preventing them from being precisely quantified. If unmetabolised enniatin B were to be measured, a reference standard is commercially available (CAS: 917-13-5)[19].

As discussed for enniatins A and A1, specific assessment of enniatin B exposure in the indoor environment using WBE would require preliminary work to estimate baseline dietary exposure levels in New Zealand.

Overall, enniatin B is a promising candidate for WBE to assess indoor mycotoxin exposure. Monitoring for a dominant phase I metabolite (eg, M7 (*N*-demethylated enniatin B) detected in 94% of samples (Rodríguez-Carrasco et al 2018)) may eliminate the problem of non-human sources but would require assessment of its stability and detectability in wastewater, and a reference standard to be reliably quantifiable. Preliminary work would also be needed to establish baseline dietary exposure levels.

[19] https://www.sigmaaldrich.com/NZ/en/product/sigma/e5411 Accessed 8 September 2022

Table 11 Occurrence of enniatin B and its phase I metabolites in human urine

Compound/Group	Incidence (%)	Range (ng/mL)	Mean[a] (ng/mL)
Enn B	83.7	0.006–0.391	0.016
M1	43.7	0.003–0.073	0.006
M2	67.0	0.002–0.070	0.011
M3	64.3	0.002–0.087	0.012
M4	*n.d.*	–	–
M5	*n.d.*	–	–
Monooxygenated group (M1-M5)	87.7	0.005–0.107	0.033
M6	11.7	0.004–0.012	0.007
M7	93.7	0.002–0.102	0.021
N-demethylated group (M6-M7)	96.3	0.002–0.102	0.020
M8	4.0	0.007–0.552	0.077
M9	*n.d.*	–	–
M10	1.7	0.001–0.105	0.006
M11	1.0	0.006–0.015	0.009
M12	0.3	0.011	0.011
Dioxygenated group (M8-M12)	6.7	0.001–0.552	0.051

[a] Mean values are based in positive samples only.

Reproduced from Rodríguez-Carrasco et al (2018); *n* = 300.

6.2.4 Enniatin B1

Enniatin B1 was detected in eight different studies of the indoor environment assessed during preparation of this report, spanning at least seven different countries including New Zealand. Matrices in which it was detected included mouldy indoor building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15). Five biomonitoring studies assessing enniatin B1 in urine were identified. However, it was only detected in three of these studies with incidences of detection ranging from 43 – 94%. The detected concentrations ranged from < LOQ – 18 ng/mL (De Ruyck et al 2020, Escrivá et al 2017, Rodríguez-Carrasco et al 2020). Again, like enniatins A, A1 and B, the low concentrations of enniatin B1 detected in these biomonitoring studies may be due to preferential excretion of unmetabolised enniatin B1 in faeces or due to extensive metabolism of the parent compound. In the rat study detailed above, the enniatin B1 concentration in urine was below the limit of quantification whereas it was detected in faeces with an average concentration of 33 ng per gram of faeces (Escrivá et al 2015). Enniatin B1 and a range of its phase I metabolites have been reported in human urine (Rodríguez-Carrasco et al 2020) (Table 12). In this study, enniatin B1 was detected in 94% of samples at concentrations ranging from 0.007 – 0.43 ng/mL (Rodríguez-Carrasco et al 2020).

Enniatin B1 was assessed in the Berzina et al (2022) WBE study and detected in all samples with concentrations ranging from 1.4 – 10 ng/L. Results of assessment of the stability of

enniatin B1 in wastewater by Berzina et al (2022) suggest that it is relatively stable at 4°C and -18°C for up to three weeks (Table 8).

Enniatin B1 has been reported in the same range of foodstuffs as enniatin B (Santini et al 2012), and as such, potential contribution of contaminated food waste discharged to the wastewater network on levels of enniatin B1 detected using WBE must be considered. Like enniatin B, it may be possible to use one of the enniatin B1 metabolites identified by Rodríguez-Carrasco et al (2020) as a biomarker. However, like enniatin B, there are no standards available for these metabolites, preventing accurate quantification. A commercial standard is available for unmetabolised enniatin B1 (CAS: 19914-20-6)[20]. Preliminary studies would be required to assess the stability and detectability of the enniatin B1 metabolites in wastewater. As discussed for the other enniatins, specific assessment of enniatin B1 exposure in the indoor environment using WBE would also require preliminary work to estimate baseline levels of dietary exposure in New Zealand.

Table 12 Occurrence of enniatin B1 and its phase I metabolites in human urine

Compound/Group	Incidence (%)	Range (ng/mL)	Mean [a] (ng/mL)
Parent Compound			
Enn B1	94.3	0.007–0.429	0.069
Enn B1 Biotransformation Products			
M1	5.3	0.007–0.177	0.035
Demethylated and hydroxylated (M1)	5.3	0.007–0.177	0.035
M2	11.0	0.006–0.019	0.010
M3	50.0	0.005–0.076	0.023
M4	*18.0*	0.002–0.143	0.025
M5	77.3	0.006–0.186	0.047
Hydroxylated group (M2–M5)	78.0	0.006–0.233	0.069
M6	40.0	0.012–1.511	0.105
M7	30.7	0.008–0.510	0.085
M8	48.0	0.042–1.310	0.128
Carbonylated group (M6–M8)	66.0	0.012–1.763	0.196
M9	*0.7*	0.019–0.045	0.032
M10	21.0	0.008–0.241	0.047
M11	14.0	0.002–0.451	0.053
Carboxylated group (M9–M11)	26.3	0.008–0.656	0.066

[a] Mean values are based in positive samples only.

Reproduced from Rodríguez-Carrasco et al (2020); *n* = 300.

Overall, similar to enniatin B, enniatin B1 is a promising candidate for WBE to assess indoor mycotoxin exposure. Ideally, one of its phase I metabolites should be used as the biomarker to eliminate the problem of non-human sources, but this would require a reference standard to be reliably quantifiable. Preliminary work is also needed to estimate baseline dietary exposure levels.

[20] https://www.sigmaaldrich.com/NZ/en/product/sigma/e5286 Accessed 8 September 2022

6.2.5 Beauvericin

Beauvericin was detected in ten different studies of the indoor environment assessed during preparation of this report, spanning at least seven different countries including New Zealand. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15). Three biomonitoring studies assessing beauvericin in urine were identified. However, none reported detection of beauvericin in any of the samples (Escrivá et al 2017, Kyei et al 2022, Warensjö Lemming et al 2020).

A recent 2022 study identified 15 different metabolites in addition to unmetabolised beauvericin when mouse, rat, dog, monkey and human liver microsomes were incubated with beauvericin (Yuan et al 2022) (Table 13). Thirteen of these metabolites were produced by human liver microsomes. However, a study in mice found that beauvericin was undetectable in urine after intraperitoneal injection (Rodríguez-Carrasco et al 2016).

Beauvericin was assessed in the Berzina et al (2022) WBE study but only detected in 14% of the samples, with all being below the limit of quantification. An assessment of the stability of beauvericin in wastewater by Berzina et al (2022) showed that its levels reduced by 35% after two weeks at 4°C and by around 40% after 2 – 3 weeks at -18°C (Table 8).

Table 13 Summary of beauvericin and its metabolites in mouse, rat, dog, monkey and human liver microsomes

Code	$[M+H]^+$ *m/z*	RT [b] (min)	Formula	Mouse	Rat	Dog	Monkey	Human	Metabolic Pathways [c]
M1	1107.4960	7.61	$C_{55}H_{74}N_6O_{16}S$	+	+	+	+	-	Oxygenation, and glutathione conjugation (P + O + GSH)
M2	935.4095	7.91	$C_{48}H_{62}N_4O_{13}S$	+	+	+	+	+	Di-oxygenation, cysteine conjugation (P + 2O + Cys)
M3	832.4009	8.46	$C_{45}H_{57}N_3O_{12}$	+	+	+	+	-	Tri-oxygenation (P + 3O)
M4	832.4003	8.63	$C_{45}H_{57}N_3O_{12}$	+	+	+	+	+	Tri-oxygenation (P + 3O)
M5	832.4011	8.87	$C_{45}H_{57}N_3O_{12}$	+	+	+	+	+	Tri-oxygenation (P + 3O)
M6	802.3901	9.66	$C_{44}H_{55}N_3O_{11}$	+	+	+	+	+	Di-oxygenation, *N*-demethylation (P + 2O–CH_2)
M7	816.4054	9.73	$C_{45}H_{57}N_3O_{11}$	+	+	+	+	+	Di-oxygenation (P + 2O)
M8	800.4105	9.95	$C_{45}H_{57}N_3O_{10}$	+	-	+	+	+	Mono-oxygenation (P + O)
M9	816.4052	10.28	$C_{45}H_{57}N_3O_{11}$	+	+	+	+	+	Di-oxygenation (P + 2O)
M10	800.4106	10.90	$C_{45}H_{57}N_3O_{10}$	+	+	+	+	+	Mono-oxygenation (P + O)
M11	816.4055	10.93	$C_{45}H_{57}N_3O_{11}$	+	+	+	+	+	Di-oxygenation (P + 2O)
M12	800.4113	10.97	$C_{45}H_{57}N_3O_{10}$	-	-	-	+	+	Mono-oxygenation (P + O)
M13	800.4108	11.30	$C_{45}H_{57}N_3O_{10}$	+	+	+	+	+	Mono-oxygenation (P + O)
M14	800.4105	11.99	$C_{45}H_{57}N_3O_{10}$	+	+	+	+	+	Mono-oxygenation (P + O)
M15	770.4002	12.75	$C_{44}H_{55}N_3O_9$	+	+	+	+	+	*N*-demethylation (P – CH_2)
BEA	784.4156	13.01	$C_{45}H_{57}N_3O_9$	+	+	+	+	+	NA

Reproduced from Yuan et al (2022).

Beauvericin has been detected in wastewater treatment plant effluents in Switzerland (Schenzel et al 2012, Schenzel et al 2010) and the US (Kolpin et al 2014). In the 2010 Swiss study, beauvericin was present in all four samples collected but below the limit of quantification (Schenzel et al 2010). In a follow-on 2012 study, beauvericin was detected in 4/6 effluent samples collected from the same wastewater treatment plant, with concentrations at the limit of detection (3.4 ng/L) (Schenzel et al 2012). Beauvericin has also been detected in effluents from three wastewater treatment plants in New York, with an incidence of detection of 67% (2/3 samples) (Kolpin et al 2014). However, the concentrations were very low, with both samples noted as detected but not quantified (Kolpin et al 2014).

Beauvericin has been reported in a range of foods including wheat, oats, barley and maize (EFSA 2014), and mouldy garlic (Sulyok et al 2007), vegetables, jam, bread and fruit (Sulyok et al 2010). As such, the potential contribution of contaminated food waste discharged to the wastewater network on levels of beauvericin detected using WBE must be considered. It may be possible to measure one of the metabolites of beauvericin identified by Yuan et al (2022) (Table 13) to eliminate the problem of non-human sources adding to levels measured using WBE. However, preliminary work would be required to confirm the metabolite is excreted and is stable and detectable in wastewater. Similar to enniatins B and B1, there are unlikely to be standards available for these metabolites. A commercial standard is available for unmetabolised beauvericin (CAS: 26048-05-5)[21]. Specific assessment of beauvericin exposure in the indoor environment using WBE would also require preliminary work to estimate baseline levels of dietary exposure in New Zealand.

Overall, beauvericin is a promising candidate for WBE. It has been shown to be detectable in wastewater, albeit at very low concentrations. Sub-catchment screening focused on small populations where considerable indoor exposure is suspected may allow for greater detection due to less dilution from unaffected individuals. It may also be possible to screen for a metabolite of beauvericin, eliminating the problem of non-human sources present in wastewater, but this would require substantial preliminary work.

6.3 TRICHOTHECENES

6.3.1 Verrucarol

Verrucarol was detected in 11 different studies of the indoor environment assessed during preparation of this report, spanning six different countries. Matrices in which it was detected included mouldy/water-damaged building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15). One biomonitoring study assessing verrucarol in urine was identified. In this study verrucarol was only detected in 2.1% of samples and at very low concentration (0.0016 – 0.0047 ng/L) (De Ruyck et al 2020).

Limited additional information on the excretion of verrucarol was identified during preparation of this report. Two studies of excretion in dogs were identified (Barel et al 1990, Barel et al 1994). In the first of these studies, less than 1% of an intravenous administered dose of verrucarol was found to be excreted unchanged in urine (Barel et al 1990). A follow-on study reported that around 1% of an intravenous administered dose was excreted unchanged in urine, whereas 75% of the dose was excreted as verrucaryl glucuronides and verrucaryl sulfates (Barel et al 1994). As noted above for sterigmatocystin, glucuronides are not good candidates for monitoring using WBE (Banks et al 2018). Although, as for sterigmatocystin, if the conjugates were transformed back to the parent form in wastewater (eg, deconjugated), they could be measured as such.

Verrucarol was also detected in urine and faeces from Iranian soldiers exposed to trichothecene mycotoxins during a gas attack (Heyndrickx et al 1984).

[21] https://www.sigmaaldrich.com/NZ/en/product/sigma/b7510 Accessed 8 September 2022

Very little information was also found on the potential occurrence of verrucarol in foodstuffs, and a recent review noted that data on the prevalence of verrucarol in food and feed is very limited (Chen et al 2020).

Overall, there is currently insufficient information on the metabolism, excretion and detectability/stability of verrucarol or its metabolites in wastewater for WBE to be employed. Additionally, it is unclear whether macrocyclic trichothecenes present in wastewater would be hydrolysed in-sewer to verrucarol, confounding detected levels. Substantial preliminary work is required before WBE could be used to assess indoor exposure to verrucarol.

6.3.2 Trichodermol

Trichodermol was detected in six different studies of the indoor environment assessed during preparation of this report, spanning four different countries. Matrices in which it was detected included water-damaged building materials and settled dust (Appendix Table 15).

No information on the excretion of trichodermol or its presence in foods was identified during preparation of this report. As such, there is currently insufficient information for WBE to be employed to assess exposure to trichodermol in the indoor environment.

6.3.3 Satratoxin G

Satratoxin G was detected in ten different studies of the indoor environment assessed during preparation of this report, spanning five different countries, including in New Zealand. Matrices in which it was detected included mouldy/water-damaged building materials, floor dust and indoor air (Appendix Table 15).

The kinetics of satratoxin G excretion following intranasal exposure has been studied in mice (Amuzie et al 2010). This study showed that satratoxin G is excreted in both urine and faeces but cumulatively accounted for less than 0.3% of the administered dose (Amuzie et al 2010). However, the authors noted that the method used to detect satratoxin G was by ELISA and it is possible that the satratoxin G was metabolised to products that did not react with the ELISA (Amuzie et al 2010). Alternatively, the intranasally administered satratoxin G may have been poorly absorbed by the nasal mucosa, thereby limiting the amount excreted. No published information on human excretion of satratoxin G was identified during preparation of this report. However, a company marketing a urine-based assay for satratoxin G exposure was identified[22]. No further information on excretion of satratoxin G was identified.

No information on presence of satratoxin G in wastewater was identified during preparation of this report. However, satratoxin G is known to be able to be hydrolysed to verrucarol (Bloom et al 2007, Clarke et al 2021). As such, the potential hydrolysis of satratoxin G to verrucarol in-sewer should be considered.

A 2009 study assessed the presence of satratoxin G in a range of cereals and cereal products (Gottschalk et al 2009). However, it was not detected in any of the samples tested.

[22] https://www.vibrant-wellness.com/tests/mycotoxins/#1527504422796-ce510636-2662 Accessed 19 September 2022

Overall, there is currently insufficient information on the excretion and metabolism of satratoxin G for WBE to be employed.

6.3.4 Satratoxin H

Satratoxin H was detected in ten different studies of the indoor environment assessed during preparation of this report, spanning five different countries. Matrices in which it was detected included mouldy/water-damaged indoor building materials and indoor air (Appendix Table 15).

No published information on human excretion of satratoxin H was identified during preparation of this report, but a company marketing a urine-based assay for satratoxin H exposure was identified[23]. No reports identifying satratoxin H in foodstuffs were identified, although it was detected in bedding straw (Harrach et al 1983), suggesting it could be present in products from animals that consumed infected straw. Similar to satratoxin G, it was also screened for but not detected in a range of cereal and cereal products (Gottschalk et al 2009). As noted above, *S. chartarum* has been shown to produce satratoxin H when artificially inoculated onto wheat, oats, barley, corn and rice (Stack & Eppley 1980),

Overall, there is currently insufficient information on the excretion and metabolism of satratoxin H for WBE to be employed.

6.4 ALTERNARIOL AND ALTERNARIOL MONOMETHYL ETHER

6.4.1 Alternariol

Alternariol was detected in seven different studies of the indoor environment assessed during preparation of this report, spanning at least five different countries including New Zealand. Matrices in which it was detected included mouldy/water-damaged indoor building materials and floor dust (Appendix Table 15). Eight biomonitoring studies assessing alternariol in urine were identified (Appendix Table 19). However, it was only detected in six of these studies, with incidences of detection ranging from 7 to 52% and concentrations ranging from below the limit of quantification to 46 ng/mL (Table 6) (De Ruyck et al 2020, Fan et al 2021, Martins et al 2019, Qiao et al 2022, Qiao et al 2020, Šarkanj et al 2018). This low detection may be due to preferential excretion of alternariol in faeces rather than urine, as a study in rats found that more than 89% of an oral dose of alternariol was excreted in faeces, mostly unmetabolised, whilst only 2.8% was excreted in urine (Puntscher et al 2019). Two alternariol metabolites were also identified - alternariol-3-sulfate (0.8% of dose, excreted in urine) and 4-hydroxy alternariol (1% of dose, excreted in faeces) (Puntscher et al 2019).

A 2020 Chinese human biomonitoring study found that incubation of urine samples with β-glucuronidase/arylsulfatase increased the amount of alternariol detected, and noted that 90 – 95% of alternariol was present as a conjugated form in urine (Qiao et al 2020).

[23] https://www.vibrant-wellness.com/tests/mycotoxins/#1527504422796-ce510636-2662 Accessed 19 September 2022

Overall, alternariol is a promising candidate for WBE as it has already been detected in human urinary biomonitoring studies and may also be present in faeces, as in rats. If alternariol is preferentially excreted in faeces in humans, then testing wastewater which contains both faeces and urine would likely allow for better detection than was seen in the urinary biomonitoring studies. However, alternariol has been found in a variety of foodstuffs including maize, rice bran (Siri-Anusornsak et al 2022), tomato products (puree, juice, ketchup, peeled tomatoes), edible oils and wheat (reviewed by Ostry (2008)), and mouldy chestnuts, apples, jam and red wine (Sulyok et al 2007). As such, the potential contribution of food waste discharged to the wastewater network on levels detected using WBE must be considered. It may be possible to instead measure one of the metabolites of alternariol (eg, alternariol-3-sulfate or 4-hydroxy alternariol) to overcome this problem. However, in rats these are excreted at much lower levels than unmetabolised alternariol so may be challenging to detect. If unmetabolised alternariol were to be measured, a commercial standard is available to allow quantification (CAS: 641-38-3)[24]. Regardless of whether alternariol or one of its metabolites were to be measured, preliminary work would be required to assess its stability and detectability in wastewater. Additionally, given alternariol has been found in foodstuffs, specific assessment of indoor exposure would first require estimation of baseline dietary exposure levels. Dietary exposure estimates for alternariol have been derived overseas (EFSA (2011) and reviewed by Cressey and Pearson (2014a)). In the Mycotoxin Surveillance Program risk assessment for *Alternaria* toxins, it was noted that "*Alternaria* toxins are almost certainly present in the New Zealand food supply. However, concentrations in foods and levels of dietary exposure are currently unknown" (Cressey & Pearson 2014a).

6.4.2 Alternariol monomethyl ether

Alternariol monomethyl ether was detected in eight different studies of the indoor environment assessed during preparation of this report, spanning at least six different countries including New Zealand. Matrices in which it was detected included mouldy/water-damaged indoor building materials and floor and settled dust (Appendix Table 15). Seven biomonitoring studies assessing alternariol monomethyl ether in urine were identified (Appendix Table 19). However, it was only detected in four of these studies. The incidence of detection varied dramatically from 7 to 96%. The detected concentrations ranged from below the limit of quantification to 22 ng/mL (Table 6) (De Ruyck et al 2020, Fan et al 2021, Qiao et al 2022, Qiao et al 2020). Similar to alternariol, this low detection may be due to preferential excretion of alternariol monomethyl ether in faeces rather than urine as in rats > 85% of an oral dose was excreted in faeces within 24 hours, whereas only 2.6% was excreted in urine (Puntscher et al 2019). Most of the alternariol monomethyl ether excreted in faeces was unmetabolised. Small amounts of alternariol monomethyl ether-3-sulfate was detected in urine (0.6% of dose after 24 hours) and 4-hydroxy alternariol monomethyl ether was identified at the limit of detection in faeces (Puntscher et al 2019).

Overall, alternariol monomethyl ether is a promising candidate for WBE as like alternariol it has already been detected in biomonitoring studies and may also be present in faeces. However, alternariol monomethyl ether has been found in similar foodstuffs to alternariol,

[24] https://www.sigmaaldrich.com/NZ/en/product/sigma/a1312 Accessed 8 September 2022

including tomato products (puree, juice, ketchup, peeled tomatoes) and edible oils (reviewed by Ostry (2008)), and mouldy chestnuts, apples, and red wine (Sulyok et al 2007). As such, the potential contribution of food waste discharged to the wastewater network on measured alternariol monomethyl ether levels must be considered. It may be possible to instead measure one its metabolites, although, in rats these are excreted at even lower levels than the alternariol metabolites so will likely be difficult to reliably detect in wastewater. If unmetabolised alternariol monomethyl ether were to be measured, a commercial standard is available (CAS: 23452-05-3)[25]. Preliminary work would be required to assess the stability and detectability of alternariol monomethyl ether (or its metabolite) in wastewater. Additionally, given alternariol monomethyl ether has been detected in foods, specific assessment of indoor exposure requires estimation of baseline dietary exposure levels. Similar to alternariol, dietary exposure estimates have been derived for alternariol monomethyl ether internationally (EFSA (2011) and reviewed by Cressey and Pearson (2014a)), but dietary exposure in New Zealand is unknown (Cressey & Pearson 2014a).

6.5 OTHERS

6.5.1 Emodin

Emodin was detected in nine different studies of the indoor environment assessed during preparation of this report, spanning at least six different countries including New Zealand. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15).

After oral administration of ^{14}C-emodin to rats, 22% of the dose was excreted in urine in 72 hours and 68% was excreted in faeces in 120 hours (Bachmann & Schlatter 1981). Most (~ 70%) of the ^{14}C-emodin excreted in urine was free anthraquinones (emodin and emodic acid), 14% was conjugated emodin or emodic acid, and 2% was unidentified (Bachmann & Schlatter 1981). In comparison, only around 40% of the dose excreted in faeces was free anthraquinones (emodin and emodic acid), less than 2% was conjugated emodin or emodic acid and 22% was unidentified (Bachmann & Schlatter 1981).

A separate rat study found that after oral administration of rhubarb extract or rhubarb total free anthraquinone oral colon-specific drug delivery granules (RTFAOCDD-GN) known to contain emodin, 0.9 and 7.6% of the emodin was excreted in urine, and 0.9 and 5.1% was excreted in faeces for rhubarb extract and RTFAOCDD-GN respectively (Zhang et al 2015). A third rat study found that after oral administration of rhubarb extract, the excretion rate of emodin was 0.6% and 64% in urine and faeces respectively (Zhao et al 2021).

No information on the excretion of emodin by humans was identified during preparation of this report. If it is excreted in urine and faeces as in rats, then it may be measurable using WBE. However, this would require considerable preliminary work to determine if it is excreted and if it is stable and detectable in wastewater. Additionally, emodin has been detected in a range of foods including maize, millet, (Warth et al 2012a), mouldy bread, fruit, vegetables, jam, nuts (Sulyok et al 2010), chestnuts and red rice (Sulyok et al 2007). As such, the potential contribution from non-human sources to the levels of emodin measured

[25] https://www.sigmaaldrich.com/NZ/en/product/sigma/a3171 Accessed 8 September 2022

using WBE would need to be considered. It may be possible to instead measure a metabolite of emodin to overcome this problem, but as no information was identified on metabolism of emodin by humans, this would require substantial preliminary work. Specific assessment of indoor exposure to emodin using WBE would also require estimation of baseline dietary exposure levels, as detailed above. As such, emodin is currently unsuitable as a candidate for WBE to assess indoor mycotoxin exposure.

6.5.2 Stachybotrylactam

Stachybotrylactam was detected in eight different studies of the indoor environment assessed during preparation of this report, spanning at least seven different countries. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15).

No toxicokinetic information from animals or humans was identified during preparation of this report.

Stachybotrylactam has been found in maize and rice bran (Siri-Anusornsak et al 2022), and in straw (animal feed) (Ulrich et al 2021). As discussed above, this means the potential contribution of contaminated food waste discharged to the wastewater network on levels detected using WBE would need to be considered. However, no information on excretion of stachybotrylactam was identified during preparation of this report. As such, it is not currently a suitable candidate for WBE.

6.5.3 Roquefortine C

Roquefortine C was detected in eight different studies of the indoor environment assessed during preparation of this report, spanning at least seven different countries. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, floor and settled dust, and indoor air (Appendix Table 15). Three biomonitoring studies assessing roquefortine C in urine were identified (Appendix Table 19). However, it was only detected in two of these studies and with low incidence of detection (4 – 7%; Table 6). The detected concentrations were in the range < LOQ – 40 ng/mL (Al-Jaal et al 2021, De Ruyck et al 2020). It is possible that this low detection is due to preferential excretion of roquefortine C in faeces rather than urine, as a study in rats found that around 56% of an oral dose of ^{14}C-roquefortine C, and 56 – 85% of an intraperitoneal dose was excreted in faeces, whereas only around 5% of the oral dose and 4% of the intraperitoneal dose was excreted in urine (Laws & Mantle 1987). Approximately one third of the roquefortine excreted in faeces was unchanged, whilst the remainder was more polar roquefortine metabolites (Laws & Mantle 1987).

Overall, roquefortine C is a promising candidate for WBE as it has already been detected in human urine using biomonitoring and may also potentially be present in faeces (as for rats). However, roquefortine C has been reported in a range of foods including blue-vein cheese (Maragos 2022), maize (Janić Hajnal et al 2020) and mouldy bread, fruit, vegetables, nuts, cheese and jam (Sulyok et al 2010). As such, the potential contribution of food waste discharged to the wastewater network on levels of roquefortine C detected using WBE must be considered. It may be preferable to identify a metabolite of roquefortine C to use as a

biomarker instead to overcome this problem. If unmetabolised roquefortine C were to be measured, a commercial standard is available to allow for quantification (CAS: 58735-64-1)[26]. Regardless of whether unmetabolised roquefortine C or a metabolite were measured, preliminary work would be required to determine its stability and detectability in wastewater. Additionally, given roquefortine C is known to be present in foodstuffs, specific assessment of indoor exposure would require estimation of baseline dietary exposure levels.

6.5.4 Meleagrin

Meleagrin was detected in eight different studies of the indoor environment assessed during preparation of this report, spanning at least six different countries. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15).

No toxicokinetic information from animals or humans was identified during preparation of this report.

No information on the excretion of meleagrin, or presence in wastewater, could be identified during preparation of this report. As such, this mycotoxin is not currently a suitable candidate for WBE as more information is required, particularly confirming whether meleagrin or a suitable metabolite is excreted in urine and/or faeces.

6.5.5 Physcion

Physcion was detected in six different studies of the indoor environment assessed during preparation of this report, spanning four different countries including New Zealand. Matrices in which it was detected included moisture-damaged building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15).

Excretion of physcion has been studied in rats orally administered rhubarb extract known to contain this toxin. In one study, the excretion rate of physcion after oral administration of rhubarb extract was 0.13% for urinary excretion and 81% for faecal excretion (Zhao et al 2021). In a separate study, rats were orally administered either rhubarb extract or rhubarb total free anthraquinone oral colon-specific drug delivery granules (RTFAOCDD-GN) known to contain physcion (Zhang et al 2015). After administration 0.5 and 3.8% of the physcion was excreted in urine, and 0.7 and 6.3% was excreted in faeces for rhubarb extract and RTFAOCDD-GN respectively (Zhang et al 2015). A third study in rats found that within 72 hours, 13 – 21% of an oral dose of physcion was excreted in faeces, mostly unmetabolised, and only approximately 0.2% of the dose was excreted in urine (reviewed by Li et al (2020)).

Although no information of excretion of physcion by humans was identified during preparation of this report, if it is excreted in a similar manner to rats then it may be measurable using WBE. However, this would require considerable preliminary work to determine if it is indeed excreted in human faeces and/or urine, and to assess its stability and detectability in wastewater. Additionally, physcion has been reported in a variety of foods including rhubarb (Zhang et al 2015, Zhao et al 2021), maize (poultry feed) (Warth et

[26] https://www.sigmaaldrich.com/NZ/en/product/sigma/sml0406 Accessed 8 September 2022

al 2012a), lettuce and beans (Mueller et al 1999), and is “a major bioactive ingredient in the traditional Chinese medicine Radix et Rhizoma Rhei” (Pang et al 2016). As such, the potential contribution from non-human sources to the levels of physcion measured using WBE would need to be considered. Specific assessment of indoor exposure to physcion would also require estimation of baseline dietary exposure levels, as detailed above.

6.5.6 Penicillic acid

Penicillic acid was detected in five different studies of the indoor environment assessed during preparation of this report, spanning four different countries. Matrices in which it was detected included materials from water-damaged buildings and floor and settled dust (Appendix Table 15).

A study in rats using ^{14}C-penicillic acid has shown that the majority of the administered dose is excreted in urine, with 82% excreted after 7 days (Park et al 1980). A separate study in mice using ^{14}C-penicillic acid found that over 90% of an intravenous dose and approximately 60% of an intraperitoneal dose was excreted in urine (Chan et al 1984). However, the authors note that “essentially no unchanged PA [penicillic acid] was detected in the urine”, with the ^{14}C-penicillic acid being excreted as glutathione or glucuronide conjugates (Chan et al 1982, Chan et al 1984). Only a small amount (5 – 6%) of the dose was excreted in faeces (Chan et al 1984). No additional information on excretion was identified during preparation of this report.

Overall, although evidence from mice and rat studies have shown that penicillic acid metabolites are excreted in urine, considerable work would still be required to identify a suitable human metabolite to use as a biomarker for WBE. This would include identification of human urinary penicillic acid metabolites (assuming unmetabolised penicillic acid is not excreted in urine, as in mice) and assessment of their stability and detectability in wastewater. Additionally, given penicillic acid has been reported in a range of foods, including maize, dried beans and several different fruits (reviewed by Cressey and Pearson (2014a)), specific assessment of indoor exposure would first require estimation of baseline dietary exposure levels.

6.5.7 Chaetoglobosin A

Chaetoglobosin A was detected in six different studies of the indoor environment assessed during preparation of this report, spanning at least four different countries. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, floor dust and indoor air (Appendix Table 15).

No published evidence of chaetoglobosin being present in urine or faeces was identified during preparation of this report. However, several companies marketing urine-based tests for chaetoglobosin A were identified[27,28,29], suggesting markers for chaetoglobosin A in urine

[27] https://healthmatters.io/understand-blood-test-results/chaetoglobosin Accessed 19 September 2022
[28] https://fxmed.co.nz/product/functional-testing/the-great-plains-lab/gpl-mycotox/ Accessed 19 September 2022
[29] https://www.vibrant-wellness.com/tests/mycotoxins/#1527504422796-ce510636-2662 Accessed 19 September 2022

are available. However, substantial work would be required to determine the excretion profile of chaetoglobosin A in urine and/or faeces, or to identify another chaetoglobosin A metabolite suitable for WBE. As such, chaetoglobosin A is currently unsuitable for monitoring using WBE.

6.5.8 Griseofulvin

Griseofulvin was detected in seven different studies of the indoor environment assessed during preparation of this report, spanning six different countries. Matrices in which it was detected included mouldy surface swabs, and floor and settled dust (Appendix Table 15).

Given griseofulvin has been used as a pharmaceutical, several studies have assessed its absorption and excretion in humans. A 1961 study found that about 11% of an oral dose of griseofulvin (17 mg/kg bw, taken daily for 30 days) was excreted in urine as 6-demethyl griseofulvin, with no unchanged griseofulvin detected (Barnes & Boothroyd 1961). A separate study, published in 1971, found that around 65% of both an oral and intravenous dose of griseofulvin was excreted in urine as 6-desmethylgriseofulvin or its glucuronide, with no unchanged griseofulvin detected (Chiou & Riegelman 1971). This urinary excretion rate was considerably higher than that reported by Barnes and Boothroyd (1961), however, the authors noted that "the urinary excretion rate of weak acids like 6-demethyl griseofulvin…may depend upon the pH of the urine", with a higher pH favouring excretion (Chiou & Riegelman 1971). A third study, published in 1973 found that about 50% of an oral dose was excreted in urine and 36% in faeces within five days (Lin et al 1973). The main urinary metabolite was 6-desmethyl griseofulvin, representing 84% of the total amount excreted in urine, with 55% of this being free 6-desmethyl griseofulvin and 45% being its glucuronide (Lin et al 1973). Only around 0.1% of the dose was excreted unchanged in urine, and around 1% was excreted as 4-desmethyl griseofulvin (Lin et al 1973).

Overall, griseofulvin is a promising candidate for WBE. Results from the studies detailed above suggest that the best candidate biomarker for WBE would be 6-desmethyl griseofulvin, given glucuronides are unsuitable for WBE (Banks et al 2018). Screening for this metabolite will also avoid the complication of non-human sources of the parent griseofulvin present in wastewater, given it is known to be present in a range of foods including maize (Janić Hajnal et al 2020, Matumba et al 2015), folio millet, sesame (Ezekiel et al 2014), and mouldy chestnuts and tomatoes (Sulyok et al 2007). Additionally, there may be contribution to the wastewater network from individuals using griseofulvin as a pharmaceutical. 6-desmethylgriseofulvin is commercially available (CAS 20168-88-1)[30], allowing quantification. However, no information on the presence or stability of 6-desmethylgriseofulvin in wastewater was identified during preparation of this report. As such, preliminary studies would be needed to establish the stability of this compound in wastewater and to determine whether it can be reliably detected. Additionally, given griseofulvin is known to be present in foods, determination of indoor exposure using WBE would first require estimation of a baseline dietary exposure level, using an approach such as urinary biomonitoring.

[30] https://www.scbt.com/p/6-o-demethyl-griseofulvin-20168-88-1 Accessed 19 September 2022

6.5.9 3-Nitropropionic acid

3-Nitropropionic acid was detected in five different studies of the indoor environment assessed during preparation of this report, spanning four different countries including New Zealand. Matrices in which it was detected included mouldy surface swabs, and floor and settled dust (Appendix Table 15).

Despite being associated with significant health effects in humans, little information was identified on the excretion of 3-nitropropionic acid. 3-Nitropropionic acid has been detected in bovine urine after intravenous administration, accumulating to high levels (1 – 3mM) (Majak & McDiarmid 1990). As such, the authors suggested that "urine analysis for NPA [3-nitropropionic acid] could well be used for diagnostic purposes" (Majak & McDiarmid 1990). The excretion rate of 3-nitropropionic acid in cattle was noted to be "much more rapid" than that seen in another study undertaken in rats, with most of the administered dose excreted within 3 hours (Majak & McDiarmid 1990, Matsumoto et al 1961). In the rat study, rats were intraperitoneally administered 3-nitropropionic acid which was then excreted in urine for up to 36 hours, with between 1.8 – 2.4% of the administered dose excreted in the urine (Matsumoto et al 1961). It was noted the difference in excretion rate between cattle and rats may have been due to differences in dosage and method of administration (Majak & McDiarmid 1990). 3-Nitropropionic acid has been detected in the urine of an acutely intoxicated child (using untargeted metabolomics) (Bendiksen Skogvold et al 2022), indicating it is also excreted via this route in humans. No information on potential excretion in faeces, or presence in wastewater, was identified.

3-Nitropropionic acid has been found in a variety of foodstuffs including maize (Matumba et al 2015), fonio millet and sesame (Ezekiel et al 2012), and mouldy vegetables (Sulyok et al 2010). As for the other mycotoxins discussed above, this means the potential contribution of contaminated food waste discharged to the wastewater network on levels of 3-nitropropionic acid detected using WBE would need to be considered. Additionally, to specifically assess indoor exposure to 3-nitropropionic acid, baseline dietary exposure levels in New Zealand would need to be estimated.

Overall, it may be possible to use WBE to assess 3-nitropropionic acid exposure in the indoor environment. However, considerable preliminary work is required to fully assess excretion of 3-nitropropionic acid, or its metabolites, in humans, and the stability and detectability of these compounds in wastewater. If 3-nitropropionic acid were to be measured, a commercial standard is available to facilitate quantification (CAS: 504-88-1)[31].

6.5.10 Equisetin

Equisetin was detected in five different studies of the indoor environment assessed during preparation of this report, spanning at least four different countries. Matrices in which it was detected included mouldy/water-damaged indoor building materials, mouldy surface swabs, and floor and settled dust (Appendix Table 15).

No toxicokinetic information from animals or humans was identified during preparation of this report.

[31] https://www.sigmaaldrich.com/NZ/en/product/sigma/n5636 Accessed 8 September 2022

Equisetin has been found in a variety of foodstuffs including maize (Matumba et al 2015), wheat (Scarpino et al 2015) and fonio millet (Ezekiel et al 2012). As for the other mycotoxins discussed above, this means the potential contribution of contaminated food waste discharged to the wastewater network on levels of equisetin detected using WBE would need to be considered. However, no information on excretion of equisetin by any animal, or presence in wastewater, was identified during preparation of this report. As such, this mycotoxin is not currently a suitable candidate for WBE as more information is required, particularly confirming whether equisetin and/or its metabolites are excreted in urine and faeces.

7. SUMMARY

Exposure to mycotoxins present in the indoor environment, particularly damp or water-damaged buildings, may pose a human health hazard. However, due to a lack of biomonitoring information, little is known about the level of human exposure to mycotoxins in the indoor environment in New Zealand. Recently, WBE has been used as a powerful population-level tool to monitor exposure to a wide range of substances, including mycotoxins. However, the two published WBE studies assessing mycotoxin exposure mainly focused on dietary exposure.

The aim of this report was to assess whether WBE could be used to specifically assess human exposure to mycotoxins present in the indoor environment.

The first step in this assessment involved a review of published and grey literature to identify which mycotoxins have been identified in indoor environments. This resulted in generation of a list of more than 140 different fungal secondary metabolites detected in various matrices including indoor building materials, surface swabs, settled and floor dust, and indoor air. Twenty-three of these metabolites, which were all classified as mycotoxins, were chosen for further assessment of their suitability for WBE based on their detection in five or more different studies. A high-level overview of the potential human health hazard posed by these mycotoxins was then conducted, which determined that 22 of these likely pose some risk to human health, primarily based on information from animal studies.

These 22 mycotoxins were then assessed to determine whether they are excreted in urine and/or faeces, and therefore are potentially detectable in wastewater. Initially, a review of international urinary biomonitoring studies was performed to determine if exposure to any of these candidates had already been assessed using urine. The candidates were then further assessed to identify any additional information on their excretion and/or metabolism in humans, and potential presence in food.

Overall, ten promising candidate mycotoxins were identified: sterigmatocystin, enniatins A, A1, B and B1, beauvericin, alternariol and alternariol monomethyl ether, roquefortine C and griseofulvin, as summarised in Table 14.

Sterigmatocystin has been detected by urinary biomonitoring, albeit at low levels. This may be due to preferential excretion in faeces rather than urine, as is seen in rats. Monitoring of wastewater which contains both urine and faeces would therefore likely increase detection levels. In vervet monkeys and cattle, sterigmatocystin is predominantly excreted as a glucuronide conjugate. This has not been confirmed in humans, but a glucuronide conjugate would be undesirable as a biomarker for WBE as glucuronides have been shown to rapidly transform in wastewater and are therefore unsuitable for WBE. It may be possible to screen for unchanged sterigmatocystin, as done in the urinary biomonitoring studies. However, given it has been detected in several different foodstuffs, contribution of non-human sources present in wastewater to levels detected using WBE would need to be considered. Additionally, the stability and detectability of sterigmatocystin in wastewater would also need to be confirmed. Targeting of WBE to sub-catchments where substantial exposure is suspected (eg, areas with known water-damage) may enhance detection due to less dilution by unexposed individuals.

Enniatins A and A1 have both been assessed by urinary biomonitoring, but only A1 was detected, and at low levels. Similar to sterigmatocystin this may be due to preferential excretion in faeces. Both enniatins A and A1 were detected using WBE in Latvia at low levels. Unchanged enniatin A and A1 could be used as a biomarker for WBE, but as for sterigmatocystin, the contribution of non-human sources to levels detected by WBE would need to be considered, Targeting of WBE to sub-catchments with suspected high exposure levels may allow for enhanced detection.

Enniatins B and B1 have both been detected in urinary biomonitoring at low levels. Similar to enniatins A and A1, this may be due to preferential excretion in faeces. Both have also been detected using WBE in Latvia. Previous work identified several human urinary metabolites of both enniatin B and B1 which could be used as biomarkers instead of unchanged enniatin B/B1, removing the complication of non-human sources present in wastewater from contributing to levels measured using WBE. However, this would require preliminary work to confirm these metabolites are both stable and detectable in wastewater.

Beauvericin has also been a target of urinary biomonitoring but was not detected. It was, however, detected by WBE in Latvia, albeit at very low levels. Given beauvericin has been detected in a range of foodstuffs, if unchanged beauvericin were to be used as a biomarker for WBE the contribution of non-human sources to levels detected using WBE would need to be considered. As discussed above, targeting to sub-catchments with suspected high exposure may enhance detection levels.

The *Alternaria* toxins, alternariol and alternariol monomethyl ether, have both been detected at low levels in urinary biomonitoring. This may be due to preferential excretion in faeces rather than urine, as in rats. However, both are mostly excreted unchanged so the contribution of non-human sources present in wastewater to detected levels would need to be considered. Additionally, the stability and detectability of these mycotoxins in wastewater would also need to be confirmed.

Roquefortine C has also been detected in urinary biomonitoring but like many of the mycotoxins detailed above is preferentially excreted in faeces in rats. Unchanged roquefortine C could be used as the biomarker as in rats one third of an administered dose is excreted unchanged in faeces. However, as noted above, non-human sources present in wastewater would need to be considered. It may be possible to screen for a human metabolite, but this would require substantial preliminary work to identify human urinary metabolites. Whether unchanged roquefortine C, or a metabolite, were chosen as the biomarker, its stability and detectability in wastewater would first need to be confirmed.

Considerable information is available for the excretion and metabolism of griseofulvin given it has been used as a human pharmaceutical. It is known to mostly be excreted in urine as 6-desmethyl griseofulvin and its glucuronide, with some additional faecal excretion. Given glucuronides are unsuitable biomarkers for WBE, it would be preferable to use 6-desmethyl griseofulvin as the biomarker, although its stability and detectability in wastewater would first need to be confirmed.

All ten of these mycotoxins have been reported in various foodstuffs. As such, specific assessment of indoor exposure to these mycotoxins using WBE would first require estimation of baseline dietary exposure levels which could then be subtracted from measured levels to estimate indoor exposure. This could be done using urinary

biomonitoring. However, as noted above, many of these mycotoxins are preferentially excreted in faeces in animals. If they are also preferentially excreted in faeces in humans, urinary biomonitoring may not provide a true representation of exposure. Faecal biomonitoring may instead be a more suitable option. Additionally, estimation of baseline dietary exposure levels may be complex due to factors such as cultural differences in food consumption and seasonal differences in dietary mycotoxin exposure.

For the remaining twelve mycotoxins there was insufficient information available on human excretion and/or metabolism for WBE to currently be viable. For verrucarol, although it has been detected at very low levels in a urinary biomonitoring study, and in urine and faeces from Iranian soldiers exposed to trichothecene mycotoxins during a gas attack, no additional information on excretion or metabolism in humans was available. Similarly, 3-nitropropionic acid has been detected in the urine of an acutely intoxicated child and in animal urine, but no additional information on its human excretion or metabolism was identified. As such, considerable preliminary work would be required prior to WBE being considered for these two mycotoxins.

For satratoxin G, emodin, physcion and penicillic acid some information on excretion in animals was identified. However, except for identification of a commercial urine-based test for satratoxin G exposure, no information on human excretion or metabolism was identified. As such, considerable work would be require to confirm if these mycotoxins are indeed excreted in human urine and/or faeces, and whether they are excreted unchanged or as metabolites. For trichodermol, stachybotrylactam, meleagrin and equisetin no information on metabolism or excretion was identified. For satratoxin H and chaetoglobosin A the only information pertaining to excretion was identification of commercial urine-based tests for assessing exposure to these mycotoxins.

Overall, this report has identified ten promising candidate mycotoxins for assessment of exposure to mycotoxins in the indoor environment using WBE. However, before any WBE studies could be initiated preliminary work is required for all ten. In particular, baseline dietary exposure to these mycotoxins in New Zealand needs to be estimated to allow indoor exposure to be estimated. Additionally, for those mycotoxins, or their metabolites, which have not already been assessed using WBE their stability and detectability in wastewater must also be confirmed.

Table 14 Summary of suitability of candidate mycotoxins for WBE

Mycotoxin	Detected in human urinary biomonitoring or WBE	Suitable alternative human excretion data identified	Promising candidate for WBE?
Sterigmatocystin	✓		**YES**
Enniatin A	✓		**YES**
Enniatin A1	✓		**YES**
Enniatin B	✓		**YES**
Enniatin B1	✓		**YES**
Beauvericin	✓		**YES**
Verrucarol	✓^	✗	NO
Trichodermol	NA	✗	NO
Satratoxin G	NA	✗	NO
Satratoxin H	NA	✗	NO
Alternariol	✓		**YES**
Alternariol monomethyl ether	✓		**YES**
Emodin	NA	✗	NO
Stachybotrylactam	NA	✗	NO
Roquefortine C	✓		**YES**
Meleagrin	NA	✗	NO
Physcion	NA	✗	NO
Penicillic acid	NA	✗	NO
Chaetoglobosin A	NA	✗	NO
Griseofulvin	NA	✓	**YES**
3-nitropropionic acid	NA	✗*	NO
Equisetin	NA	✗	NO

NA, not assessed. *Detected in urine from a single patient, using untargeted metabolomics. ^Detected only in a single biomonitoring study, in 2.1% of samples all at very low concentrations.

GLOSSARY

3AcDON	3-Acetyldeoxynivalenol
15AcDON	15-Acetyldeoxynivalenol
ADD	Average daily dose
AME	Alternariol monomethyl ether
AOH	Alternariol
BHK cells	Baby hamster kidney cells
BRANZ	Building Research Association of New Zealand
bw	Body weight
CONTAM panel	Contaminants in the food chain panel
CIRS	Chronic inflammatory response syndrome
DOM-1	Deepoxy-deoxynivalenol
DON	Deoxynivalenol
EFCOVAL project	European Food Consumption Validation project
EFSA	European Food Safety Authority
GC-MS	Gas chromatography-mass spectrometry
GC-MS/MS	Gas chromatography-tandem mass spectrometry
HITEA	Health effects of Indoor pollutants: integrating microbial, Toxicological and Epidemiological Approaches
HPLC-MSMS	High performance liquid chromatography-tandem mass spectrometry
HRC	Health Research Council
IARC	International Agency for Research on Cancer
IC_{50}	Half the maximal inhibitory concentration
JECFA	Joint FAO/WHO Expert Committee on Food Additives
LC-MS/MS	Liquid chromatography-tandem mass spectrometry
LC-Q-TOF-MS	Liquid chromatography quadrupole time-of-flight mass spectrometry
LD_{50}	Lethal dose required to kill 50% of the test subjects
LOD	Limit of detection
LOQ	Limit of quantification

MIC	Minimum inhibitory concentration
MPI	Ministry for Primary Industries
NIV	Nivalenol
NOAEL	No observed adverse effects level
NTP	National Toxicology Program
PMTDI	Provisional maximum tolerable daily intake
QTOF	Quadrupole time-of-flight
RTFAOCDD-GN	Rhubarb total free anthraquinone oral colon-specific drug delivery granules
STE	Sterigmatocystin
TDI	Tolerable daily intake
TLC	Thin layer chromatography
TTC	Threshold of toxicological concern
WBE	Wastewater-based epidemiology
WHO	World Health Organization
ZEN	Zearalenone

APPENDIX

Table 15 Mycotoxins/fungal secondary metabolites identified in indoor environment samples

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Trichothecenes	Verrucarol	14 ng/g sample	LC-MS/MS	Mouldy building materials	Finland	Tuomi et al (2000)
		7.7 – 600 ng/g	GC-MS/MS	Building materials from damp/water-damaged buildings	Sweden	Bloom et al (2007)
		8.8 – 17,000 ng/g	GC-MS/MS	Water-damaged building materials	Sweden	Bloom et al (2009b)
		Not quantified	GC-MS	*S. chartarum*-infested building materials[32]	Chicago, USA	Croft et al (1986)
		5.3 ng/m^2 index	GC-MS/MS	Mouldy surface swabs from schools	Spain	Peitzsch et al (2012)
		830 ng/m^2 index, reference and non-categorised	GC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		0.6 – 18 ng/g dust	GC-MS/MS	Floor dust from water-damaged homes	New Orleans, USA	Bloom et al (2009a)
		19 – 43 ng/g	GC-MS/MS	Settled dust from damp/water-damaged buildings	Sweden	Bloom et al (2007)
		0.6 – 1.7 ng/g	GC-MS/MS	Settled dust from water-damaged buildings	Sweden	Bloom et al (2009b)
		2.6 ng/m^2	GC-MS/MS	Settled dust from schools	Malaysia	Norbäck et al (2016)

[32] Referred to by its former name *Stachybotrys atra*.

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Trichothecenes	Verrucarol	0.6 – 37 ng/g	GC-MS/MS	Floor dust from water-damaged buildings	USA	Saito et al (2016)
		10 ng/m^2 reference	GC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		66 ng/m^2 reference	GC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		40 ng/m^2 index	GC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		13,000 ng/m^2 <0.025 – 1.0 ng/g	GC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
	Verrucarin B	Not quantified	TLC	*S. chartarum*-infested building materials[33]	Chicago, USA	Croft et al (1986)
	Verrucarin J	600,000 ng/m^2	LC-MS/MS	Artificially inoculated wallpaper		Aleksic et al (2017)
		80 ng	LC-MS/MS	Aerosols from artificially inoculated wallpaper		Aleksic et al (2017)
		Not quantified	TLC	*S. chartarum*-infested building materials[34]	Chicago, USA	Croft et al (1986)
		Not quantified	LC-MS/MS	Mouldy indoor building materials	Germany	Gottschalk et al (2006)
	Satratoxin G	2.5 – 770 ng/g sample	LC-MS/MS	Mouldy building materials	Finland	Tuomi et al (2000)
		Not quantified	LC-MS/MS	Water-damaged building materials	Sweden	Bloom et al (2009b)
		1,300,000 – 11,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)

[33] Referred to by its former name *Stachybotrys atra*.
[34] Referred to by its former name *Stachybotrys atra*.

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Trichothecenes	Satratoxin G	1,400,000 ng/m^2	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		0.25 ng/m^3	LC-MS/MS	Indoor air from water-damaged building with known *Stachybotrys* contamination	Germany	Gottschalk et al (2008)
		Not quantified	LC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		Not quantified	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		4,400,000 – 22,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
		7,100,000 ng/m^2	LC-MS/MS	Artificially inoculated wallpaper		Aleksic et al (2017)
		100 ng	LC-MS/MS	Aerosols from artificially inoculated wallpaper		Aleksic et al (2017)
		Not quantified	LC-MS/MS	Building materials from damp/water-damaged buildings	Sweden	Bloom et al (2007)
		180,000 – 97,000,000 ng/m^2	LC-MS/MS	Mouldy indoor building materials	Germany	Gottschalk et al (2006)
	Satratoxin H	80 - 93 ng/g sample	LC-MS/MS	Mouldy building materials	Finland	Tuomi et al (2000)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Trichothecenes	Satratoxin H	Not quantified	LC-MS/MS	Water-damaged building materials	Sweden	Bloom et al (2009b)
		0.43 ng/m^3	LC-MS/MS	Indoor air from water-damaged building with known *Stachybotrys* contamination	Germany	Gottschalk et al (2008)
		Not quantified	LC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
		2,300,000 – 13,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
		14,000,000 ng/m^2	LC-MS/MS	Artificially inoculated wallpaper		Aleksic et al (2017)
		1000 ng	LC-MS/MS	Aerosols from artificially inoculated wallpaper		Aleksic et al (2017)
		Not quantified	TLC	*S. chartarum*-infested building materials[35]	Chicago, USA	Croft et al (1986)
		630,000 – 6,200,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		1,400,000 – 1,800,000 ng/m^2	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		Not quantified	LC-MS/MS	Building materials from damp/water-damaged buildings	Sweden	Bloom et al (2007)

[35] Referred to by its former name *Stachybotrys atra*.

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Trichothecenes	Satratoxin H	Not quantified	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		200,000 – 120,000,000 ng/m^2	LC-MS/MS	Mouldy indoor building materials	Germany	Gottschalk et al (2006)
	Satratoxin F	Not quantified	LC-MS/MS	Mouldy indoor building materials	Germany	Gottschalk et al (2006)
	Satratoxin (mixture of G and H)	<1,000 – 17,000 ng/g sample	HPLC	Indoor building materials from a water-damaged day care centre	Finland	Andersson et al (1997)
	Trichodermol	0.9 – 8,700 ng/g	GC-MS/MS	Water-damaged building materials	Sweden	Bloom et al (2009b)
		3.4 – 18,000 ng/g	GC-MS/MS	Building materials from damp/water-damaged buildings	Sweden	Bloom et al (2007)
		2.4 – 3.4 ng/g	GC-MS/MS	Settled dust from damp/water-damaged buildings	Sweden	Bloom et al (2007)
		22 ng/m^2 reference	GC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		60 ng/m^2 reference	GC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		31 ng/m^2 index 5.2 ng/m^2 non-categorised	GC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		< 0.025 ng/g	GC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
	Trichodermin	63 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Spain	Peitzsch et al (2012)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Trichothecenes	Trichodermin	9,600 ng/m^2 index, reference and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
	Trichoverrin A	Not quantified	HPLC	*S. chartarum*-infested building materials[36]	Chicago, USA	Croft et al (1986)
	Trichoverrin B	Not quantified	HPLC	*S. chartarum*-infested building materials[37]	Chicago, USA	Croft et al (1986)
	Roridin E	0.0031 – 0.082 ng/m^3	LC-MS/MS	Indoor air from mouldy water-damaged building	Belgium	Polizzi et al (2009)
		Not quantified	LC-MS/MS	Mouldy indoor building materials	Germany	Gottschalk et al (2006)
		64 ng/m^2	UHPLC-MS/MS	Settled dust from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		170,000 – 1,300,000 ng/m^2	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
	Roridin L2	Not quantified	LC-MS/MS	Mouldy indoor building materials	Germany	Gottschalk et al (2006)
		5,900,000 ng/m^2	LC-MS/MS	Artificially inoculated wallpaper		Aleksic et al (2017)

[36] Referred to by its former name *Stachybotrys atra*.
[37] Referred to by its former name *Stachybotrys atra*.

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Trichothecenes	Roridin L2	350,000 – 550,000 ng/m^2	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		64 ng	LC-MS/MS	Aerosols from artificially inoculated wallpaper		Aleksic et al (2017)
	Deoxynivalenol	920 ng/g sample	LC-MS/MS	Mouldy building materials	Finland	Tuomi et al (2000)
		64 – 150 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	3-acetyl-deoxynivalenol	19 – 22 ng/g sample	LC-MS/MS	Mouldy building materials	Finland	Tuomi et al (2000)
	Diacetoxyscirpenol	14 – 3,300 ng/g sample	LC-MS/MS	Mouldy building materials	Finland	Tuomi et al (2000)
	T-2 tetraol	110,000 ng/g sample	LC-MS/MS	Mouldy building materials	Finland	Tuomi et al (2000)
	Total macrocyclic trichothecenes	28,000,000 ng/m^2	LC-MS/MS	Artificially inoculated wallpaper		Aleksic et al (2017)
		1,300 ng	LC-MS/MS	Aerosols from artificially inoculated wallpaper		Aleksic et al (2017)
		0.62 ppb mouldy 0.29 ppb control	ELISA	Indoor air from flooded homes	France	Charpin-Kadouch et al (2006)
		22 ppb mouldy 0.31 ppb control	ELISA	Floor dust from flooded homes	France	Charpin-Kadouch et al (2006)
		40 ppb mouldy 0.25 ppb control	ELISA	Mouldy wall surfaces from flooded homes	France	Charpin-Kadouch et al (2006)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Trichothecenes	Total macrocyclic trichothecenes	3.1 – 1,400 trichothecene equivalents/m^3 mouldy 2.2 – 120 trichothecene equivalents/m^3 control	ELISA	Indoor air from mould-contaminated buildings	Texas, USA	Brasel et al (2005)
Cyclohexadepsipeptides	Enniatin A	0.01 – 6.9 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		78 – 80 ng/g dust	LC-MS/MS	House dust	Germany	Lindemann et al (2022)
		1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		0.7 – 220 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
		51 ng/m^2 index 1.5 ng/m^2 reference 0.5 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		80 ng/m^2 index 1.5 ng/m^2 reference	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		1.1 – 2.6 ng/g unrepaired 0.36 – 2.1 ng/g repaired 0.83 – 1.3 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		0.83 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Cyclohexadepsipeptides	Enniatin A	2,100 ng/m^2 index and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
	Enniatin A1	0.06 – 34 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		1.4 – 190 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
		15 ng/m^2 index 37 ng/m^2 reference	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		130 ng/m^2 index 1.9 ng/m^2 reference 4.0 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		86 ng/m^2 index 11 ng/m^2 reference	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		1.6 – 9.3 ng/g unrepaired 1.6 – 5.6 ng/g repaired 1.6 – 4.6 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		0.86 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		1.0 ng/g	LC-MS/MS	Floor dust from severely moisture damaged buildings	Finland	Täubel et al (2011)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Cyclohexadepsipeptides	Enniatin A1	2.7 ng/g	LC-MS/MS	Dust in vacuum cleaner bags from severely moisture damaged buildings	Finland	Täubel et al (2011)
		6,500 ng/m^2 non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
	Enniatin B	0.11 – 41 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		< LOQ – 560 ng/g dust	LC-MS/MS	House dust	Germany	Lindemann et al (2022)
		0.9 – 10 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		10 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		0.3 – 8.2 ng/g unrepaired 0.38 – 3.4 ng/g repaired 0.36 – 2.7 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		6.9 ng/m^2 index 12 ng/m^2 reference 1.8 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		63 ng/m^2 index 23 ng/m^2 reference 1.0 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Cyclohexadepsipeptides	Enniatin B	170 ng/m^2 index 130 ng/m^2 reference 1.3 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		0.44 – 0.78 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		1.6 – 4.9 ng/g	LC-MS/MS	Floor dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
		0.35 – 1.1 ng/g	LC-MS/MS	Settled dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
		0.63 – 9.4 ng/g	LC-MS/MS	Dust in vacuum cleaner bags from severely moisture damaged buildings	Finland	Täubel et al (2011)
		51,000 ng/m^2 index and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		5,000 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Finland	Peitzsch et al (2012)
	Enniatin B1	0.16 – 69 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		3.8 – 42 ng/g dust	LC-MS/MS	House dust	Germany	Lindemann et al (2022)
		10 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		1.0 – 7.5 ng/g unrepaired 1.0 – 7.1 ng/g repaired 1.1 – 6.2 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Cyclohexadepsipeptides	Enniatin B1	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		24 ng/m^2 index 43 ng/m^2 reference	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		110 ng/m^2 index 3.4 ng/m^2 reference 1.4 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		340 ng/m^2 index 120 ng/m^2 reference	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		15,000 ng/m^2 index and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		10,000 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Finland	Peitzsch et al (2012)
	Enniatin B2	570 ng/m^2 non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		1.9 ng/g	LC-MS/MS	Control house dust	USA or India	Vishwanath et al (2011)
		0.6 – 2 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
	Total enniatins (A, A1, B, B1)	5,100 – 1,100,000 ng/m^2; < 3.3 ng/g	LC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
	Beauvericin	0.45 – 18 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		0.11 – 70 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Cyclohexadepsipeptides	Beauvericin	0.75 – 1.4 ng/g	LC-MS/MS	Floor dust from severely moisture damaged homes	Finland	Täubel et al (2011)
		0.67 – 1.6 ng/g	LC-MS/MS	Settled dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
		0.32 ng/g	LC-MS/MS	Dust in vacuum cleaner bags from severely moisture damaged buildings	Finland	Täubel et al (2011)
		0.7 – 1.8 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp buildings	Finland	Vishwanath et al (2011)
		3.1 ng/g	LC-MS/MS	Control house dust	USA or India	Vishwanath et al (2011)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		8.7 – 190 ng/g dust	LC-MS/MS	House dust	Germany	Lindemann et al (2022)
		3.4 ng/m^2 index 5.8 ng/m^2 reference 0.8 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		40 ng/m^2 index 96 ng/m^2 reference 0.3 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		4.2 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Cyclohexadepsipeptides	Beauvericin	19,000 – 3.1 x 10^{12} ng/m^2; <2.8 ng/g	LC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
		5000 ng/m^2 index and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
	Bassianolide	0.95 – 110 ng/g unrepaired 0.91 – 570 ng/g repaired 1.1 – 130 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Sterigmatocystin	Sterigmatocystin	630 – 7,800,000 ng/m^2	LC-MS/MS	Dust or mouldy surfaces/materials from water-damaged buildings	Belgium	Polizzi et al (2009)
		0.2 – 31,000 ng/g sample	LC-MS/MS	Mouldy building materials	Finland	Tuomi et al (2000)
		1.9 – 1,100 ng/g	LC-MS/MS	Building materials damp/water-damaged buildings	Sweden	Bloom et al (2007)
		2,000 – 20,000,000 ng/m^2	HPLC-DAD	Water-damaged building materials	Denmark	Nielsen et al (1999)
		100,000 ng/m^2 index, reference and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		1,200 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Finland	Peitzsch et al (2012)
		4.9 – 150,000 ng/g	LC-MS/MS	Water-damaged building materials	Sweden	Bloom et al (2009b)
		1.8 – 3,900 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Sterigmatocystin	Sterigmatocystin	21 – 110 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		3.8 ng/g	LC-MS/MS	Settled dust from a primary health care centre	Portugal	Viegas et al (2021)
		1.3 ng/g	LC-MS/MS	Floor dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
		16 – 28 ng/g dust	LC-MS/MS	Floor dust from water-damaged homes	New Orleans, USA	Bloom et al (2009a)
		17 ng/g	LC-MS/MS	Settled dust from damp/water-damaged building	Sweden	Bloom et al (2007)
		2.0 – 3.8 ng/g dust	LC-MS/MS	Carpet dust from mouldy and/or damp houses	Germany	Engelhart et al (2002)
		< LOQ – 3,100 ng/g dust	LC-MS/MS	House dust	Germany	Lindemann et al (2022)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		< 1 [conc] ≤ ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		1.6 – 11 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
		1.4 – 3.3 ng/g unrepaired 1.4 – 590 ng/g repaired 1.2 – 90 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Sterigmatocystin	Sterigmatocystin	1.7 ng/m^2	LC-MS/MS	Settled dust from schools	Malaysia	Norbäck et al (2016)
		50 ng/m^2 index	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		27 ng/m^2	LC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
		0.0034 – 1.8 ng/m^3	LC-MS/MS	Indoor air from mouldy water-damaged buildings	Belgium	Polizzi et al (2009)
		82 – 480 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
		110,000,000 ng/m^2	LC-MS/MS	Artificially inoculated wallpaper		Aleksic et al (2017)
		180 ng	LC-MS/MS	Aerosols from artificially inoculated wallpaper		Aleksic et al (2017)
	5-methoxy-sterigmatocystin	4,000 – 8,000,000 ng/m^2	HPLC-DAD	Water-damaged building materials	Denmark	Nielsen et al (1999)
		1.4 – 53 ng/g unrepaired 0.90 – 7,700 ng/g repaired 2.4 – 6,800 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Methoxy-sterigmatocystin	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	Seco-sterigmatocystin	2.8 – 490 ng/g repaired 2.8 – 89 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Aflatoxins	Aflatoxin B1	1,100 – 3,300 ng/m^2	LC-MS/MS	Dust or mouldy surfaces/materials from water-damaged buildings	Belgium	Polizzi et al (2009)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Aflatoxins	Aflatoxin B1	0.00084 ng/g; 12 ng/m^2	LC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
		0.0024 – 0.15 ng/m^3	LC-MS/MS	Indoor air from mouldy water-damaged buildings	Belgium	Polizzi et al (2009)
		2 – 7 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
	Aflatoxin B2	190 – 2,500 ng/m^2	LC-MS/MS	Dust from mouldy water-damaged buildings	Belgium	Polizzi et al (2009)
		0.0003 – 0.021 ng/m^3	LC-MS/MS	Indoor air from mouldy water-damaged buildings	Belgium	Polizzi et al (2009)
	Aflatoxin G1	11 – 12 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
Versicolorins	Versicolorin A	3.2 – 87 ng/g repaired 84 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Versicolorin C	1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		2.4 – 11 ng/g unrepaired 5.8 – 720 ng/g repaired 19 – 630 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Anthraquinoids	Emodin	3,200 ng/m^2 index 170 ng/m^2 reference 27 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Anthraquinoids	Emodin	160 ng/m^2 index 140 ng/m^2 reference 13 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		46 ng/m^2 index 300 ng/m^2 reference 24 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		10 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		4 – 120 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
		15 ng/g	LC-MS/MS	Control house dust	USA or India	Vishwanath et al (2011)
		45 – 140 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		0.24 – 15 ng/g	LC-MS/MS	Floor dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
		84 ng/g	LC-MS/MS	Settled dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
		2.4 – 39 ng/g	LC-MS/MS	Dust in vacuum cleaner bags from severely moisture damaged buildings	Finland	Täubel et al (2011)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Anthraquinoids	Emodin	0.24 – 180 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		4,800 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Spain	Peitzsch et al (2012)
		310 ng/m^2 index and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		280 ng/m^2 index and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	Finland	Peitzsch et al (2012)
	Norsolorinic acid	[conc] < 1 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		6.4 – 18 ng/g unrepaired 18 – 81 ng/g repaired 27 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Averufin	10 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		2.3 – 8.2 ng/g unrepaired 2.4 – 720 ng/g repaired 2.4 – 230 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Averantin	[conc] < 1 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		1.4 ng/g unrepaired 0.75 – 51 ng/g repaired 1.3 – 39 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Anthraquinoids	Macrosporin A	10 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
	Macrosporin	7.7 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		120 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
	Nidurufin	9.3 – 51 ng/g repaired 42 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	Chrysophanol	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	Citreorosein	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	Endocrocin	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	Fallacinol	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Ergot alkaloids	Chanoclavine	72 ng/m^2 index and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		Not quantified	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		3.2 ng/g unrepaired 1.8 – 41 ng/g repaired 3.8 – 8.4 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		[conc] < 1 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Ergot alkaloids	Festuclavine	[conc] < 1 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		2.8 – 9.6 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		3.3 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
	Fumigaclavine (= Fumiclavine)	8.2 ng/m^2 reference	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		65 ng/m^2 index	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		810 ng/m^2 index and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		Not quantified	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
	Agroclavine	[conc] < 1 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		1.5 – 2.6 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Dihydrolysergol	2.8 ng/m^2 reference	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		7.1 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Alternaria toxins	Alternariol	10 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		13 – 400 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		35 – 41 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
		40 ng/g	LC-MS/MS	Control house dust	USA or India	Vishwanath et al (2011)
		10 – 13 ng/g unrepaired 22 – 38 ng/g repaired 25 – 400 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		38 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
	Alternariol monomethyl ether	1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		2.2 – 12 ng/g unrepaired 2.2 – 15 ng/g repaired 3.0 – 96 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		8.0 ng/g	LC-MS/MS	Control house dust	USA or India	Vishwanath et al (2011)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Alternaria toxins	Alternariol monomethyl ether	7.3 – 10 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
		13 ng/m^2 index	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		1.2 – 55 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		1.3 ng/g	LC-MS/MS	Floor dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
		7.6 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
	Altenuene	33 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		1,200 ng/g	LC-MS/MS	Floor dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
	Altertoxin	210 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
	Altersetin	37 – 1,400 ng/g repaired 22 – 860 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Tenuazonic acid	460 ng/g unrepaired 98 – 260 ng/g repaired 110 – 240 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Indole alkaloids	Roquefortine C	0.009 – 4 ng/m^3	LC-MS/MS	Indoor air from mouldy water-damaged buildings	Belgium	Polizzi et al (2009)
		1,100 – 72,000 ng/m^2	LC-MS/MS	Dust or mouldy surfaces/materials from water-damaged buildings	Belgium	Polizzi et al (2009)
		4.2 ng/m^2 reference	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		10 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		< 2.2 ng/g	LC-MS/MS	Settled dust from a primary health care centre	Portugal	Viegas et al (2021)
		40 – 120 ng/g repaired 25 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		29 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Spain	Peitzsch et al (2012)
		79,000 ng/m^2 index and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		100 – 1,100 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
		4.2 – 1,000 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
	Roquefortine D	90 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Aspergamide A	340 – 410 ng/g repaired 22 – 39 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Indole alkaloids	Brevianamide F	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		10 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
	Marcfortine A	5.1 – 24 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Notoamide derivative	1.2 – 4.1 ng/g unrepaired 3.1 – 2,200 ng/g repaired 6.8 – 540 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Oxaline	2.6 – 18 ng/g unrepaired 2.1 – 9.8 ng/g repaired 2.2 – 5.7 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Cytochalasans	Chaetoglobosin A	12,000 – 140,000,000 ng/m^2	LC-MS/MS	Mouldy indoor surfaces from water-damaged buildings	Belgium	Polizzi et al (2009)
		55 – 130,000 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		83 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		3,100 ng/g	LC-MS/MS	Floor dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
		270,000 – 25,000,000 ng/m^2	HPLC-DAD	Water-damaged building materials	Denmark	Nielsen et al (1999)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Cytochalasans	Chaetoglobosin A	14,000 – 12,000,000 ng/m^2	LC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
		0.0067 – 3.4 ng/m^3	LC-MS/MS	Indoor air from mouldy water-damaged buildings	Belgium	Polizzi et al (2009)
		2,200,000 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Finland	Peitzsch et al (2012)
		Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
	Chaetoglobosin C	1,000,000 – 4,000,000 ng/m^2	HPLC-DAD	Water-damaged building materials	Denmark	Nielsen et al (1999)
		Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
	Cytochalasin B	9.4 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
	Cytochalasin D	2.9 – 1,900 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		57 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
Imidazopyridoindole	Meleagrin	880 ng/m^2 index	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		76 ng/m^2 index 51 ng/m^2 reference 300 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		100 ng/m^2 index	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Imidazopyridoindole	Meleagrin	Not quantified	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		630 – 2,800 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		3.5 – 62,000 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		730 – 14,000 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
		Not quantified	HPLC-DAD	Water-damaged building materials	Denmark	Nielsen et al (1999)
		8,200 ng/m^2	LC-MS/MS	Mouldy surface swabs from schools	Spain	Peitzsch et al (2012)
		7,400,000 ng/m^2 index, reference and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		1,400 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Finland	Peitzsch et al (2012)
Anthracenone	Physcion	89,000 ng/m^2 index 54,000 ng/m^2 reference 1,400 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		11,000 ng/m^2 index 6,200 ng/m^2 reference 530 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		170 ng/m^2 index	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Anthracenone	Physcion	1 < [conc] < 1000 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		420 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		2,500 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Finland	Peitzsch et al (2012)
		46 – 460 ng/g	LC-MS/MS	Dust in vacuum cleaner bags from severely moisture damaged buildings	Finland	Täubel et al (2011)
		73,000 ng/m^2 index, reference and non-categorised	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
Benzodiazepine	Cyclopenol	1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		15 – 65 ng/g unrepaired 23 – 15000 ng/g repaired 28 – 12000 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Cyclopenin	1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		2.6 ng/g unrepaired 3.4 – 330 ng/g repaired 5.5 – 150 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Cyclopeptine	1 < [conc] ≤ 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Benzodiazepine	Cyclopeptine	2.4 ng/g unrepaired 1.0 – 150 ng/g repaired 4.1 – 48 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Dehydrocyclopeptine	160 ng/g repaired 60 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Asperloxine A	110 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Benzofuran	Stachybotrylactam	1 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		440 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		5,500 ng/m^2 index	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		2,000 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		1,600,000 – 10,400,000 ng/m^2	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		70 – 2,300 ng/m^2	UHPLC-MS/MS	Settled dust from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		< 33,000 – 82,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Benzofuran	Stachybotrylactam	62,000 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Spain	Peitzsch et al (2012)
		19,000,000 ng/m^2 index and reference	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
		1,300 – 2,600 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
		300,000 – 5,200,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	Stachybotrylactam isomer	1,400,000 – 9,100,000 ng/m^2	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		700 – 1,800 ng/m^2	UHPLC-MS/MS	Settled dust from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
	Stachybotrylactam acetate	7,300 – 15,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		40,000 – 310,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	Stachybotrysin B	8,400,000 – 130,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		720,000 – 330,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	Stachybotrysin C	540,000 – 6,300,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Benzofuran	Stachybotrysin C	170,000 – 25,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	Stachybonoid D	< LOQ	LC-MS/MS	House dust	Germany	Lindemann et al (2022)
		4,300,000 – 100,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		310,000 – 170,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	Stachybotrydial	1,700,000 – 5,400,000 ng/m^2	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		800 – 1,000 ng/m^2	UHPLC-MS/MS	Settled dust from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		900,000 – 77,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		1,700,000 – 120,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	Stachybotrydial acetate	100,000 – 23,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		88,000 – 47,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Benzofuran	2α-acetoxystachybotrydial acetate	2,900,000 – 110,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		330,000 – 110,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	Stachybotryamide	74 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		360,000 – 5,100,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		1,700,000 – 1,800,000 ng/m^2	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		230,000 – < 2,300,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	L-671,667 (L-671)	< LOQ – 310 ng/g dust	LC-MS/MS	House dust	Germany	Lindemann et al (2022)
		370,000 – 25,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		500,000 – 90,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
Griseofulvin	Griseofulvin	88 ng/m^2 index	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		230 ng/m^2 index	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		240 ng/m^2 reference	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		210 ng/g	LC-MS/MS	Control house dust	USA or India	Vishwanath et al (2011)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Griseofulvin	Griseofulvin	< 1.2 ng/g	LC-MS/MS	Settled dust from a primary health care centre	Portugal	Viegas et al (2021)
		[conc] < 1 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		4.3 – 250 ng/g repaired 9.6 – 500 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		500 ng/m^2	LC-MS/MS	Mouldy surface swabs from schools	Spain	Peitzsch et al (2012)
	Dehydrogriseofulvin	3.5 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Propionic acid	3-Nitropropionic acid	590 ng/m^2 index 32 ng/m^2 reference 37 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		850 ng/m^2 index 150 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
		86 – 22,000 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
		1 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		28 ng/g repaired 23 – 57 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		260 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Finland	Peitzsch et al (2012)
Quinolinone alkaloid	Viridicatin	2,600 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Quinolinone alkaloid	Viridicatin	5.1 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		98 – 720 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
		2.6 – 4.0 ng/g unrepaired 52 – 79 ng/g repaired 9.8 – 74 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Viridicatol	63 – 97 ng/g unrepaired 51 – 2,700 ng/g repaired 85 – 6,900 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	O-Methylviridicatin	0.59 – 2.7 ng/g unrepaired 1.2 – 94 ng/g repaired 0.56 – 150 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	3-methoxy-viridicatin	Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
Butyrolactone	Penicillic acid	1,400 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		6,000 ng/m^2 index	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		510 ng/m^2 non-categorised	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		1 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		320,000 ng/m^2	LC-MS/MS	Dust from school ventilation system	Finland	Hintikka et al (2009)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Butyrolactone	Penicillic acid	2,400 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
Napthalene	Equisetin	1,900 ng/m^2 index	LC-MS/MS	Settled dust from schools	The Netherlands	Peitzsch et al (2012)
		14 – 27 ng/g unrepaired 32 – 67 ng/g repaired 9.9 – 18 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		19 – 20 ng/g	LC-MS/MS	Floor dust from severely moisture damaged/damp houses	Finland	Vishwanath et al (2011)
		26 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		3.4 – 24 ng/g	LC-MS/MS	Floor dust from severely moisture damaged buildings	Finland	Täubel et al (2011)
		6.1 – 6.4 ng/g	LC-MS/MS	Dust in vacuum cleaner bags from severely moisture damaged buildings	Finland	Täubel et al (2011)
		71 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		54 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	The Netherlands	Peitzsch et al (2012)
Integracins	Integracin A	2.2 – 11 ng/g unrepaired 2.6 – 14 ng/g repaired 4.2 – 7.4 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Integracins	Integracin B	3.5 – 17 ng/g unrepaired 2.7 – 94 ng/g repaired 3.1 – 180 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Ochratoxin	Ochratoxin A	80 – 7,700 ng/m^2	LC-MS/MS	Dust from mouldy water-damaged buildings	Belgium	Polizzi et al (2009)
		1 < [conc] < 1,000 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		8.3 – 32 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		0.012 – 0.23 ng/m^3	LC-MS/MS	Indoor air from mouldy water-damaged buildings	Belgium	Polizzi et al (2009)
Peptide (Peptaibol)	Alamethicin	620 ng/m^2 index	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		320 ng/m^2 index	LC-MS/MS	Settled dust from schools	Finland	Peitzsch et al (2012)
		Not quantified	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
	Alamethicin F30	2.5 – 10 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
		1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
	Alamethicin F50	0.11 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Phthalide	Mycophenolic acid	1,800,000 ng/m^2	LC-MS/MS	Artificially inoculated wallpaper		Aleksic et al (2017)
		270 ng	LC-MS/MS	Aerosols from artificially inoculated wallpaper		Aleksic et al (2017)
		67 – 91 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		2.5 – 4.3 ng/g	LC-MS/MS	Settled dust from primary health care centres	Portugal	Viegas et al (2021)
		Not quantified	HPLC-DAD	Artificially inoculated chipboard		Nielsen et al (1999)
Citrinin	Citrinin	20 – 35,000 ng/g sample	LC-MS/MS	Mouldy building materials	Finland	Tuomi et al (2000)
		33 ng/g unrepaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		47 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
Diterpenoid	Atranone A	Not quantified	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		Not quantified	UHPLC-MS/MS	Settled dust from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Diterpenoid	Atranone B	Not quantified	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		Not quantified	UHPLC-MS/MS	Settled dust from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
	Dolabellanes	Not quantified	UHPLC-MS/MS	Mouldy surface swab from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
		Not quantified	UHPLC-MS/MS	Settled dust from a water-damaged, severely mould contaminated kindergarten	Denmark	Došen et al (2016)
	Radiclonic acid	450 ng/g repaired 170 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Meroterpenoid	Andrastin A	7.2 – 42 ng/g repaired 19 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	Chevalone C	7.5 ng/g unrepaired 5.5 – 13 ng/g repaired 2.3 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

	Citreohybridinol	2.3 – 7.3 ng/g unrepaired 3.6 – 980 ng/g repaired 3.6 – 130 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	Stachybotrychromene A	120,000 – 4,600,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		250,000 – 6,300,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	Stachybotrychromene B	< 33,000 – 9,700,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		210,000 – 8,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
	Stachybotrychromene C	360,000 – 27,000,000 ng/m^2	LC-MS/MS	Water-damaged building materials	Germany	Jagels et al (2020)
		2,000,000 – 100,000,000 ng/m^2	LC-MS/MS	Artificially inoculated building materials		Jagels et al (2020)
Xanthones	Pinselin	12 – 40 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Norlichexanthone	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	Secalonic acid D	1 < [conc] < 1000 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		45 – 190 ng/g repaired 59 – 130 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Cyclopeptide	Cycloaspeptide A	60 ng/g unrepaired 16,000 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		160 ng/g	LC-MS/MS	Control house dust	USA or India	Vishwanath et al (2011)
	Tentoxin	1 < [conc] ≤ 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Cyclopeptide	Tentoxin	180 ng/m^2 index	LC-MS/MS	Mouldy surface swabs from schools	Finland	Peitzsch et al (2012)
Diketopiperazine	Tryprostatin B	900 – 1,900 ng/g repaired 84 – 160 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	WIN-64821	730 ng/g repaired 17 – 4,200 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Dechlorogriseofulvin	17 – 200 ng/g repaired 13 – 220 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		13 ng/g	LC-MS/MS	Control house dust	USA or India	Vishwanath et al (2011)
Diketopiperazine alkaloid	Verrucofortine	Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
		1.2 ng/g unrepaired 3.9 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Epipolythiopiperazine-3,6-diones	Chaetomin	23 ng/g	LC-MS/MS	Mouldy indoor materials	Slovakia/Austria	Vishwanath et al (2009)
	Gliotoxin	0.43 – 1.1 ng/g	LC-MS/MS	Water-damaged building materials	Sweden	Bloom et al (2009b)
		960 ng/m^2	LC-MS/MS	Dust from school ventilation systems	Finland	Hintikka et al (2009)
Penicillins	Penicillin G	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
	Amoxycillin	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Sesquiterpenoid	Sydonic acid	220 ng/g repaired 280 – 300 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Hydroxysydonic acid	43 ng/g unrepaired 84 – 5,000 ng/g repaired 150 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Tetrapeptide	Apicidin	120 ng/m^2 index	LC-MS/MS	Settled dust from schools	Spain	Peitzsch et al (2012)
		1,300 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
Anthraquinoid dimer	Skyrin	1 < [conc] < 1,000 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Amino acid	Asperphenamate	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Penitrems	Penitrem A	< LOQ	LC-MS/MS	House dust	Germany	Lindemann et al (2022)
Terpene	Culmorin	1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
Pyrone	Asteltoxin	34 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Rasfonin	310 ng/g repaired 350 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Kojic acid	2,000 ng/g	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
		140 - 540 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
	Verrucosidin	Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
Napthoquinone dimer	Aurofusarin	26 – 7,500 ng/g repaired 22 – 200 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Isocoumarin/coumarin	Monocerin	1 < [conc] < 10 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		2.6 – 6.5 ng/g unrepaired 2.3 – 6.3 ng/g repaired 1.6 – 2.8 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Orlandin	Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
Dihydroxybenzaldehyde	LL-Z 1272ε (ascochlorin[38])	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Dihydroxybenzoic acid	Orsellinic acid	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Dipeptide	Trichodermamide C	76 ng/g repaired 58 – 160 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
	Asperglaucide	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Enamide	Nigragillin	Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
Fumonisin	Fumonisin B1	92 ng/g unrepaired 50 – 63 ng/g repaired 55 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Fusariotoxin	Moniliformin	1 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)

[38] https://www.ebi.ac.uk/chebi/searchId.do?chebiId=CHEBI:156219 Accessed 14 November 2022

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Hydroquinone	Flavoglaucin	14 – 480 ng/g unrepaired 47 – 340,000 ng/g repaired 16 – 57,000 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Indole dipeptide	Neoechinulin A	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Indolyl alcohol	Tryptophol	1 < [conc] < 1,000 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
Macrolactone	Brefeldin A	Not quantified	LC-MS/MS	Building materials from severely moisture damaged buildings	Finland	Täubel et al (2011)
Naphthaquinone	Viomellein	1 < [conc] < 1,000 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
		6,900 ng/g	LC-MS/MS	Artificially inoculated building materials		Gutarowska et al (2010)
Naphthopyrones	Naphtho-*γ*-pyrones	Not quantified	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
Oxalicine alkaloid	Oxalicine B	280 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Phenyl ether	Asterric acid	1 < [conc] < 100 ng/m^2	LC-MS/MS	Floor dust	Finland	Kirjavainen et al (2016)
Polycyclic aromatic polyketide	Iso-Rhodoptilometrin	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Pyrazinoquinazoline	Fumiquinazolin derivative	50 – 280 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Quinazoline	Glyantrypine	110 – 120 ng/g repaired 190 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

Mycotoxin class	Toxin	Concentration (max unless range given)	Method	Location	Country	Reference
Quinazoline alkaloid	Chrysogin	180 ng/g repaired 27 – 310 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified (chrysogine)	HPLC-DAD	Artificially inoculated building materials		Nielsen et al (1999)
Quinolone	Quinolactacin A	0.21 – 2.0 ng/g unrepaired 0.23 – 2.7 ng/g repaired 0.21 – 8.1 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
		Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Resorcyl lactone	Zearalenone	12 ng/g unrepaired 10 ng/g repaired 9.1 –60 ng/g control	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)
Tetronic acid	Aspulvinone E	Not quantified		Carpet dust	New Zealand	Plagmann et al (2021)
Classification unknown	Unugisin E	780 ng/g repaired	LC-MS/MS	House dust	Croatia	Jakšić et al (2021)

Rounded to two significant figures. Classifications based on Bräse et al (2009) and information from https://pubchem.ncbi.nlm.nih.gov/. For Peitzsch et al (2012): index, buildings with dampness and/or moisture damage; reference, buildings without dampness/moisture damage; non-categorised, buildings where it is unclear if there is moisture damage. Values reported for Charpin-Kadouch et al (2006) are mean values ± standard deviation. Values reported for Kirjavainen et al (2016) are estimates based on information in Figure 10 as no exact values were provided. Values reported for Jakšić et al (2021), are measurements from unrepaired flooded houses, repaired flooded houses and control houses, with value ranges being the minimum and maximum readings measured across summer and winter combined. For Täubel et al (2011), where a range is given, this represents the maximum concentrations across multiple sites.

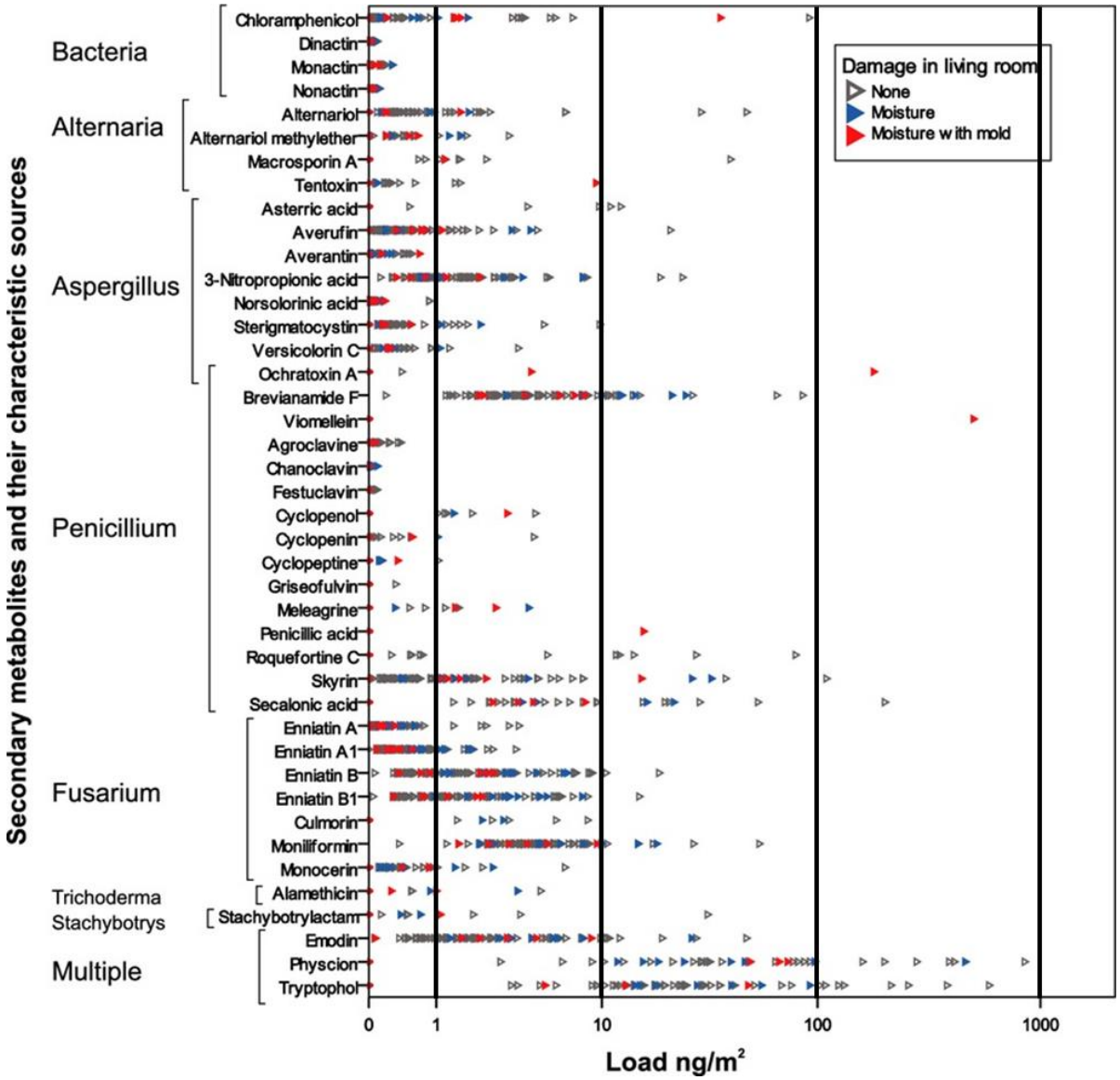


Figure 10 Abundance of microbial secondary metabolites in homes with or without moisture or mould

Adapted from Kirjavainen et al (2016).

Table 16 List of fungal and bacterial secondary metabolites screened for by Kirjavainen et al (2016)

15-Acetyldeoxynivalenol	**Asterric acid**	Cytochalasin A	Fumiquinazoline F	Neoxaline	Rugulosin
15-Hydroxyculmorin	Atpenin A5	Cytochalasin B	Fumitremorgin B	NG 012	Satratoxin G
15-Hydroxyculmoron	Aurasperon B	Cytochalasin C	Fumitremorgin C	Nidulin	Satratoxin H
15Methyl_epiFQA	Aurasperon C	Cytochalasin D	Fumonisin B1	Nidurufin	**Secalonic acid D**
16-Ketoaspergillimide	Aurasperon G	Cytochalasin E	Fumonisin B2	Nigericin	semi Vioxanthin
3-Acetyldeoxynivalenol	Aureobasidin	Cytochalasin H	Fumonisin B3	Nivalenol	Setosusin
3-AcetylDON	Aurofusarin	Cytochalasin J	Fumonisin B4	Nonactin	**Skyrin**
3-NPA	Austdiol	Daunorubicin	Fumonisin B6	Nornidulin	Spiramycin
3-O-Methylvirdicatin(M+2H)	Austocystin A	Decalonectrin	Fusaproliferin	**Norsolorinic acid**	**Stachybotrylactam**
4-Monoacetoxyscirpenol	Avenacein Y	Decarestrictin	Fusarenon-X	Nortryptoquialanine	Staurosporin
5-Hydroxyculmorin	**Averantin**	Dechlorogriseofulvin	Fusaric acid	**Ochratoxin A**	**Sterigmatocystin**
6-Aminopenicillanic acid	Averufanin	Deepoxy-deoxynivalenol	Fusarielin A	Ochratoxin alpha	Sulochrin
A 23187	**Averufin**	Deoxybrevianamid E	Fusidic acid	Ochratoxin B	Synazerol
AAL TA Toxin	Bacitracin	Deoxynivalenol	Geldanamycin	Oligomycin A	T-2 tetraol
Aflatoxin B1	Bafilomycin A1	Deoxytryptoquialanine	Geodin	Oligomycin B	T-2 toxin
Aflatoxin B2	Beauvericin	Diacetoxyscirpenol	Gibberellic acid	O-Methylsterigmatocystin	T-2 triol
Aflatoxin G1	β-Zearalenol	Dihydroergosine	Gliotoxin	Oosporin	Taxol
Aflatoxin G2	β-ZOL-Glucoside	Dihydroergotamine	**Griseofulvin**	Ophiobolin A	**Tentoxin**
Aflatoxin M1	Brefeldin A	Dihydrolysergol	HC-Toxin	Ophiobolin B	Tenuazonic acid
Aflatoxin M2	**Brevianamide F**	Dinactin	Helvolic acid	ox. Elymoclavin	Terphenyllin
Aflatrem qual	Brevicompanine B	DON Glucoside	HT-2 Toxin	ox. Luol	Terrein
Agistatin B	Butenolid	Doxorubicin	hydrolysed FB1	Oxaspirodion	Territrem B
Agistatin D	Butyrolacton II	Doxycyclin	hydrolysed Nidulin	Oxytetracyclin	Tetracycline
Agistatin E	Calphostin C	Elymoclavine	Hypothemycin	Paracelsin A	Thiolutin
Agroclavine	Cephalosporin C	Elymoclavine-Fruct	Ionomycin	Paracelsin B	TR-2 Toxin
AJ 296	Cerulenin	**Emodin**	Irgasan	Paraherquamide A	Trichodermin
Alamethicin F30	Chaetocin	**Enniatin A**	Isofusidienol	Paspalic acid	Trichostatin A
α-Zearalenol	Chaetoglobosin A	**Enniatin A1**	IsoVOL qual	Paspalin	Trypacidin
α-ZOL-Glucoside	Chaetomin	**Enniatin B**	Josamycin	Paspalinin	Tryprostatin A
Altenuene	**Chanoclavine**	**Enniatin B1**	K252a	Paspalitrem A	**Tryptophol**
Altenusin	Chetoseminudin	Enniatin B2	K252b	Paspalitrem B	Tryptoquialanine

Alternariol	Chlamydosporol	Enniatin B3	KO 143	Patulin	Tryptoquialanone
Alternariol monomethyl ether	Chloramphenicol	Equisetin	Kojic acid	Paxillin	Tryptoquivaline F
Altersolanol	Chlortetracyclin	Ergine	Lincomycin	**Penicillic acid**	Tylosin
Altertoxin-I	Chromomycin A3	Ergocornine	Lolitrem B	Penicillin G	Ustiloxin A
Altertoxin-II	Chrysophanol	Ergocorninin	Lysergol	Penicillin V	Ustiloxin B
Amoxycillin	Citreoviridin	Ergocristine	**Macrosporin A**	Penigequinolone A	Ustiloxin D
Amphotericin	Citrinin	Ergocristinine	Malformin A2	Penitrem A	Valinomycin
Andrastin A	Citromycetin	Ergocryptine	Malformin C	Pentoxyfylline	Vancomycin
Andrastin B	CJ 21058	Ergocryptinine	Marcfortine A	Pestalotin	Verrucarin A
Andrastin C	Clonostachydiol	Ergometrine	Marcfortine B	Phomopsin A	Verrucarol
Andrastin D	Cochliodinol	Ergometrinine	Marcfortine C	Phomopsin B	Verrucofortine
Anisomycin	CPA hydroly	Ergosins	**Meleagrin**	**Physcion**	Verruculogen
Anomalin A	**Culmorin**	Ergotamine	Methylsulochrin	PR Toxin	Versicolorin A
AOD-3-ol	Curvularin	Ergovalin	Methysergid	Pseurotin A	**Versicolorin C**
Apicidin	Cycloaspeptide A	Erythromycin	Mevastatin	Puromycin	**Viomellein**
Ascomycin	Cycloechinulin	**Festuclavine**	Mevinolin	Pyranonigrin	Vioxanthin
Aspercolorin	Cycloheximide	FK 506	Mithramycin C	Pyrenophorol	Viridicatin
Aspergillimide	**Cyclopenin**	Flavipucin	Mitomycin	Pyripyropene A	Wortmannin
Asperlactone	**Cyclopenol**	FS4	Monactin	Pyripyropene B	Xanthomegnin
Asperloxin A	**Cyclopeptine**	Fulvic acid	**Moniliformin**	Radicicol	Zearalenon
Asperthecin	Cyclopiazonsäure	Fumagillin	Monoacetoxyscirpenol	Rapamycin	Zearalenon-4-Sulfate
Aspinolid B	Cyclosporin A	Fumigaclavin C	**Monocerin**	**Roquefortine C**	ZON-4-Glucoside
Aspinonene	Cyclosporin C	Fumigaclavine	Mycophenolic acid	Roquefortine E	
Aspterric acid	Cyclosporin D	Fumiquinazolin A	Myriocin	Roridin A	
Aspyrone	Cyclosporin H	Fumiquinazolin D	Neosolaniol	Rubellin D	

Mycotoxins detected in living room floor dust samples by Kirjavainen et al (2016) are indicated in bold.

Table 17 List of fungal and bacterial metabolites screened for using LC-MS/MS by Vishwanath et al (2009) and Peitzsch et al (2012)

AAL TA Toxin	Avenacein Y	Deoxynivalenol	FK	Nigericin	Rubellin D
3-Acetyldeoxynivalenol	Bafilomycin A1	Deoxynivalenol-3-glucoside	Fumagillin	**3-Nitropropionic acid**	Satratoxin G
15-Acetyldeoxynivalenol	***Beauvericin***	Diacetoxyscirpenol	**Fumigaclavin A**	Nivalenol	Satratoxin H
Actinomycin D	Brefeldin A	Dihydroergosine	Fumitremorgin C	Nonactin	Secalonic acid
Aflatoxin B1	Calphostin C	Dihydroergotamine	Fumonisin B1	Ochratoxin A	***Stachybotrylactam***
Aflatoxin B2	Cephalosporin C	**Dihydrolysergol**	Fumonisin B2	Ochratoxin B	Staurosporine
Aflatoxin G1	Cerulenin	Elymoclavine	Fumonisin B3	Ochratoxin α	***Sterigmatocystin***
Aflatoxin G2	Chaetocin	Elymoclavine fructoside	Fusarenon-X	Oligomycin A	Sulochrin
Aflatoxin M1	***Chaetoglobosin A***	***Emodin***	Geldanamycin	Oligomycin B	T-2 tetraol
Aflatoxin M2	**Chanoclavine**	***Enniatin A***	Gibberellic acid	Oxaspirodion	T-2 toxin
Agroclavine	*Chetomin*	***Enniatin A1***	Gliotoxin	Oxidized elymoclavine	T-2 triol
Alamethicin F30	Chloramphenicol	***Enniatin B***	**Griseofulvin**	Oxidized luol	**Tentoxin**
Altenuene	Chromomycin A3	***Enniatin B1***	HC-toxin	Paspalin	Tenuazonic acid
Altenusin	*Citrinin*	**Enniatin B2**	HT-2 toxin	Paspalinin	Territrem B
Alternariol	Citreoviridin	Enniatin B3	Hydrolysed fumonisin	Paspalitrem A	Tetracyclin
Alternariol monomethyl ether	Cochliodinol	***Equisetin***	Ionomycin	Paspalitrem B	Trichostatin A
Altersolaniol A	Curvularin	Ergine	K252a	Patulin	Tryprostatin
Altertoxin-I	Cycloaspeptide A	Ergocornine	K252b	Paxilline	Valinomycin
2-Amino-14,16-dimethyloctadecan-3-ol	Cycloheximide	Ergocorninine	Kojic acid	**Penicillic acid**	Verrucarin A
Anisomycin	Cyclosporin A	Ergocristine	Lysergol	Penicillin G	Verrucarol
Apicidin	Cyclosporin C	Ergocristinine	Macrosporin	Penicillin A	Verruculogen
Ascomycin	Cyclosporin D	Ergocryptine	***Meleagrin***	Penitrem A	Viomellein
Asperlactone	Cyclosporin H	Ergocryptinine	Methysergide	Pentoxyfylline	*Viridicatin*
Asperloxin A	Cytochalasin A	Ergometrine	Mevinolin	**Physcion**	Wortmannin
Aspinonene	*Cytochalasin B*	Ergometrinine	Mithramycin	Pseurotin A	α-Zearalenol
Aspyrone	Cytochalasin C	Ergosine	Monactin	Puromycin	α-Zearalenol-4-glucoside
Asterric acid	*Cytochalasin D*	Ergosinine	Moniliformin	Pyripyropene A	β-Zearalenol

Atpenin A5	Cytochalasin E	Ergotamine	Monoacetoxyscirpenol	Radicicol	β-Zearalenol-4-glucoside
Aurofusarin	Cytochalasin H	Ergotaminine	Mycophenolic acid	Rapamycin	Zearalenone
Austdiol	Cytochalasin J	Ergovaline	Myriocin	***Roquefortine C***	Zearalenone-4-glucoside
Austocystin A	Deepoxydeoxynivalenol	Festuclavine	Neosolaniol	Roridin A	Zearalenone-4-sulfate

Adapted from Vishwanath et al (2009). Mycotoxins detected in school dust or swab samples by Peitzsch et al (2012) are indicated in bold. Mycotoxins detected in mouldy indoor materials by Vishwanath et al (2009) are indicated in italics. Peitzsch et al (2012) additionally screened for verrucarol and trichodermol using GC-MS/MS.

Table 18 Fungal metabolites tentatively identified in samples from water-damaged homes in Belgium by Polizzi et al (2009) using LC-Q-TOF-MS analysis

3,4-Epoxy-6-hydroxy-dolabella-7,12-dienone	Agroclavine I	Cyclo (Phe-Ser)	Dethiosecoemestrin	Fumagillin	Phaseic acid
3-Acetyldeoxynivalenol	Ascochitine	Cyclopenin	Dihydroxyaflavinine	Fumagillol	PR-toxin
5'-Hydroxyasperentin	Bis-dechlorogeodin	Cyclopeptin	Elymoclavine	Gliotoxin	Satratoxin G
Acetyl-T-2 toxin	Brevianamide A	Dechlorogriseofulvin	Epi-dechlorogriseofulvin	Italinic acid	SMTP-6*
Aflatoxin B2	Carolic acid	Deoxynivalenol	Epoxyagroclavine I	Mycophenolic acid	Vermiculin
Aflatoxin B2a	Chaetoglobosin A	Desacetylpebrolide	Equisetin	Palitantin	Verruculogen
Aflatoxin G2	Culmorin	Desmosterol	Frequentin	Penicillic acid	Vertinolide

Adapted from Polizzi et al (2009). *Triprenyl phenol derivative from *Stachybotrys microspora.*

Table 19 Summary of mycotoxin biomonitoring studies

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
China	2212	*Alternaria* toxins:	TeA, **AOH**, **AME**, TEN, ALT	TeA, **AOH**, **AME**, TEN, ALT	Qiao et al (2022)
Sweden	1096	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin: Enniatins: *Alternaria* toxins: Others:	AFB1, AFB2, AFG1, AFG2, AFM1 FB1 10-OH-OTA, OTA, 2'R-OTA, OTα DON, DON15GlcA, DOM-1, HT-2, HT-2-3-GlcA, HT-2-4-GlcA, NIV, T-2 ZAN, ZEN, ZEN14GlcA, α-ZEL, β-ZEL, α-ZELGlcA, β-ZELGlcA CIT, HO-CIT **EnA**, **EnA1**, **EnB**, **EnB1** ALT, **AME**, **AOH** BEA	 OTA DON, DON15GlcA, HT-2-3-GlcA HO-CIT	Warensjö Lemming et al (2020)
Italy, Norway, UK	635	Trichothecenes:	Free DON, total DON (free and DON3GlcA), DOM-1	Free DON, total DON, DOM-1	Brera et al (2015)
Belgium, Czech Republic, France, the Netherlands, Norway (EFCOVAL project)	600 adults	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin: Enniatins: Ergot alkaloids: *Alternaria* toxins: Others:	AFB1, AFB1-lysine adduct, AFB2, AFQ1, AFG1, AFG2, AFM1 FB1, FB2, FB3, HFB1 OTA, OTα DON, 3AcDON, 15AcDON, DOM-1, DON3Glc, DON3GlcA, DON15GlcA, NIV, T-2, HT-2, T-2 triol, T-2 tetraol, DAS, NEO ZAN, ZEN, α-ZEL, β-ZEL, ZEN14GlcA CIT **EnB1** Eco, Econ, Ecr, Ecrn, Ek, Ekn, Em, Emn, Es, Esn, Et, Etn **AOH**, **AME**, TeA **VER**, PAT, **ROQC**, **STE**	AFB1, AFB2, AFG1, AFG2, AFM1 FB1, FB2, FB3 OTA, OTα T-2, HT-2, T-2 triol, T-2 tetraol, DON, 15AcDON, DAS, DOM-1, NEO, DON3Glc, NIV α-ZEL, β-ZEL, ZAN, ZEN CIT **EnB1** Eco, Econ, Ecr, Ecrn, Ek, Ekn, Em, Emn, Es, Esn, Et, Etn **AOH**, **AME**, TeA **ROQC**, **STE, VER**	De Ruyck et al (2020)
China	599	Trichothecenes:	Total DON (free and DONGlcA)	Total DON	Wang et al (2019)

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
Qatar	559 adults	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin: Others:	AFB1, AFB2, AFG2, AFM1 FB1 OTA, OTB T-2 α-ZEL, β-ZEL CIT **ROQC**, **STE**, CPA	AFB1, AFB2, AFG2, AFM1 FB1 OTB T-2 α-ZEL, β-ZEL CIT **ROQC**, **STE**	Al-Jaal et al (2021)
Bangladesh	439 pregnant women	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin: Enniatins: *Alternaria* toxins: Others:	AFB1, AFB2, AFM1, AFG1, AFG2 FB1, FB2 OTA, OH-OTA, 2'R-OTA, OTα DON, DON3GlcA, DON15GlcA, HT-2, HT-2-3GlcA, HT-2-4GlcA, T-2 ZEN, ZAN, ZAN14GlcA, ZEN14GlcA, α-ZEL14GlcA, β-ZEL14GlcA, ZEN14-sulfate CIT, HO-CIT **EnB**, **EnB1**, **EnA**, **EnA1** **AOH**, **AME**, ALT **BEA**	AFB2, AFM1 FB1 OTA DON, DON3GlcA, DON15GlcA ZEN14-sulfate CIT, HO-CIT	Kyei et al (2022)
Belgium	155 children 239 adults	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin:	AFB1, AFB2, AFG1, AFG2, AFM1 FB1, FB2, FB3, HFB1 OTA, OTα T-2, HT-2, DON, DON3GlcA, DON15GlcA, DOM-1, DOMGlcA, 3AcDON, 3AcDON15GlcA, 15AcDON, 15AcDON3GlcA, DAS, FUSX ZEN, ZEN14GlcA, α-ZEL, α-ZEL7GlcA, α-ZEL14GlcA, β-ZEL, β-ZEL14GlcA CIT, HO-CIT	 OTA DON, DON3GlcA, DON15GlcA, DOMGlcA α-ZEL, β-ZEL14GlcA CIT, HO-CIT	Heyndrickx et al (2015)
Sweden	326 adults	Trichothecenes:	Total DON (free and DONGlcA)	Total DON	Wallin et al (2013)
China	301	Zearalenones:	ZEN, α-ZEL, β-ZEL, α-ZAL, β-ZAL, ZAN	ZEN, α-ZEL, β-ZEL, ZAN	Li et al (2018)
UK	300 adults	Trichothecenes:	Total DON (free and DONGlcA)	Total DON	Turner et al (2008)

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
Italy	300	Enniatins:	**EnB**, EnB metabolites	**EnB**, EnB metabolites	Rodríguez-Carrasco et al (2018)
Italy	300	Enniatins:	**EnB1**, EnB1 metabolites	**EnB1**, EnB1 metabolites	Rodríguez-Carrasco et al (2020)
Italy	300	Trichothecenes:	T-2, NEO, 4-deAc-NEO, 3'-OH-T-2, T-2 triol, T-2-3GlcA, HT-2-3GlcA, HT-2-4GlcA, T-2-3Glc, NEO3Glc, HT-2, HT-2-3Glc, 3'OH-T-2-3Glc, 4'OH-T-2-3Glc	NEO, 3'-OH-T-2, T-2 triol, T-2-3GlcA, HT-2-3GlcA, HT-2-4GlcA, T-2-3GlcA, NEO3Glc, HT-2, HT-2-3Glc, 3'OH-T-2-3Glc, 4'OH-T-2-3Glc,	Narváez et al (2021)
Sweden	250 adults 50 children	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones:	AFM1 FB1, FB2 OTA DON, DOM-1, NIV ZEN, α-ZEL, β-ZEL	AFM1 FB1, FB2 OTA DON, DOM-1, NIV ZEN, α-ZEL, β-ZEL	Mitropoulou et al (2018)
Pakistan	292	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones:	AFM1 FB1 OTA DON, DOM-1 ZEN, α-ZEL, β-ZEL	AFM1 FB1 OTA DON, DOM-1 ZEN, α-ZEL, β-ZEL	Xia et al (2022)
Germany, Haiti and Bangladesh	50 Germany 142 Haiti 95 Bangladesh	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin: Enniatins:	AFB1, AFB2, AFG1, AFG2, AFM1 FB1 OTA, OTα DON, T-2, HT-2, HT-2-4-GlcA, DONGlcA ZAN, ZAN14GlcA, ZEN, α-ZEL, ZEN14GlcA, α-ZEL14GlcA, β-ZEL, β-ZEL14GlcA HO-CIT **EnB**	AFM1 FB1 OTA DON, DONGlcA α-ZEL HO-CIT **EnB**	Gerding et al (2015)
China	269 adults	*Alternaria* toxins:	**AOH**, **AME**, ALT, TeA, TEN	**AOH**, **AME**, TeA, TEN	Fan et al (2021)

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
Pakistan	264	Aflatoxins: Trichothecenes:	AFM1 Total DON (free and DONGlcA)	AFM1 Total DON	Xia et al (2020)
Sweden	252 adults	Fumonisins: Ochratoxins: Trichothecenes: Zearalenones:	FB1, FB2 OTA DON, DOM-1, NIV ZEN, α-ZEL, β-ZEL	FB1, FB2 OTA DON, DOM-1, NIV ZEN, α-ZEL, β-ZEL	Wallin et al (2015)
Turkey	233 adults	Ochratoxins:	OTA	OTA	Akdemir et al (2010)
China	227 adults	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones:	AFM1, AFM2, AFB1, AFB2, AFG1, AFG2 FB1 OTA, OTα DON, DON3GlcA, DON15GlcA, 3AcDON, 15AcDON, T-2, HT-2, FUS-X ZEN, ZEN14GlcA, α-ZEL, β-ZEL, α-ZAL, β-ZAL	AFM1 FB1 OTA DON, DON3GlcA, DON15GlcA ZEN, ZEN14GlcA	Huang et al (2021)
Cameroon	220 children	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin:	AFB1, AFB1–N7Gua, AFM1 FB1, HFB1 OTA, OTα, 4-OH OTA DON, DON3Glc, DOM-1, T-2, HT-2 ZEN, α-ZEL, β-ZEL, ZENGlc CIT	AFM1 FB1 OTA DON ZEN, α-ZEL, β-ZEL	Njumbe Ediage et al (2013)
Czech Republic	205	Aflatoxins:	AFM1	AFM1	Ostry et al (2005)
China	199	Zearalenones:	ZEN, ZAN, α-ZEL, β-ZEL, α-ZAL, β-ZAL (all glucuronidase treated to give total levels – free and GlcA)	ZEN, α-ZEL, β-ZEL	Zhang et al (2020)
USA	184 adults	Aflatoxins:	AFM1	AFM1	Johnson et al (2010)
Cameroon	175 adults (83% HIV+)	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones:	AFM1 FB1, FB2 OTA DON, DON15GlcA, DON3GlcA, DOM-1, NIV, T-2, HT-2 ZEN, α-ZEL, β-ZEL, ZEN14GlcA	AFM1 FB1, FB2 OTA DON, DON15GlcA, DON3GlcA, NIV ZEN, ZEN14GlcA, α-ZEL	Abia et al (2013)

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
USA	163 girls	Zearalenones:	ZEN, α-ZEL, β-ZEL, α-ZAL, β-ZAL, ZAN	ZEN, α-ZEL, β-ZEL, α-ZAL, β-ZAL, ZAN	Bandera et al (2011)
Portugal	155 adults	Ochratoxins:	OTA	OTA	Duarte et al (2010)
Bangladesh	154 children	Ochratoxins: Citrinin:	OTA, OTα CIT, HO-CIT	OTA, OTα CIT, HO-CIT	Ali and Degen (2020)
Bangladesh	154 children	Aflatoxins: Trichothecenes:	AFM1 DON, DOM-1 (only assessed in 120 samples)	AFM1 DON	Ali et al (2020)
Tanzania	148 children	Fumonisins:	FB1	FB1	Shirima et al (2013)
China	135	*Alternaria* toxins:	TeA, ALT, **AOH**, **AME**, TEN	TeA, ALT, **AOH**, **AME**, TEN	Qiao et al (2020)
Nigeria	120	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin: *Alternaria* toxins:	AFM1 FB1 OTA DON, DOM-1, NIV ZEN, α-ZEL, β-ZEL CIT, HO-CIT **AOH**	AFM1 FB1 OTA DON, NIV ZEN, α-ZEL, β-ZEL CIT, HO-CIT **AOH**	Šarkanj et al (2018)^
Bangladesh	116 (54 pregnant women)	Aflatoxins:	AFM1	AFM1	Ali et al (2017a)
Brazil	113 adults	Aflatoxins:	AFM1, AFB1-N7-Gua	AFM1	Jager et al (2016)
Germany, Bangladesh	62 Bangladesh 50 Germany	Trichothecenes:	DON, DOM-1	DON, DOM-1	Ali et al (2016)
USA	112 with chronic fatigue syndrome	Aflatoxins: Ochratoxins: Trichothecenes:	Combined aflatoxins OTA Combined macrocyclic trichothecenes	Combined aflatoxins OTA Combined macrocyclic trichothecenes	Brewer et al (2013)

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
Germany	101 adults	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin: Enniatins:	AFB1, AFB2, AFG2, AFM1 FB1, FB2 OTA, OTα DON, DON3GlcA, T-2, HT-2, HT-2-4-GlcA ZAN, ZEN, ZEN14GlcA, ZAN14GlcA, α-ZEL, α-ZEL14GlcA, β-ZEL, β-ZEL14GlcA HO-CIT **EnB**	 T-2, DON, DONGlcA* ZEN14GlcA HO-CIT **EnB**	Gerding et al (2014)
Egypt, Guinea	50 children each	Aflatoxins:	AFM1, AFB1, AFB2, AFG1, AFG2	AFM1, AFB1, AFB2, AFG1, AFG2	Polychronaki et al (2008)
Uganda	50 children with Nodding syndrome 50 controls	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones:	AFM1 FB1, FB2 OTA T-2, HT-2 ZEN, α-ZEL, β-ZEL	AFM1 T-2 α-ZEL	Duringer et al (2021)
Portugal	95 adults	Ochratoxins:	OTA	OTA	Duarte et al (2012)
Portugal	94 adults	Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin: *Alternaria* toxins: Others:	FB1, FB2, FB3, HFB1 OTA, OTα DON, DOM-1, DON3Glc, 3AcDON, 15AcDON, DON3GlcA, DON15GlcA, NIV, FusX, DAS, NEO, T-2, HT-2, T-2 triol, T-2 tetraol ZEN, ZAN, α-ZEL, β-ZEL, α-ZEL-GlcA, β-ZEL-GlcA, α-ZAL, β-ZAL, ZEN14GlcA, ZEN14Sulf CIT **AOH**, **AME** **ROQC**, PAT, **STE**	FB1 OTA DON, DOM-1, DON3Glc, DON3GlcA, DON15GlcA ZEN, ZEN14GlcA, α-ZEL CIT **AOH**	Martins et al (2019)
Tanzania	94 children	Fumonisins:	FB1	FB1	Chen et al (2018)
Ghana	91 adults	Aflatoxins:	AFM1	AFM1	Jolly et al (2006)

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
Hungary	88	Ochratoxins:	OTA	OTA	Fazekas et al (2005)
France	76 male farmers	Trichothecenes:	DON, DOM-1	DON, DOM-1	Turner et al (2010a)
Spain	72 adults	Ochratoxins:	OTA, OTα	OTA, OTα	Coronel et al (2011)
Croatia	63	Ochratoxins:	OTA	OTA	Domijan et al (2009)
Portugal and Spain	30 Portugal 31 Spain	Ochratoxins:	OTA	OTA	Manique et al (2008)
Portugal	60 adults	Ochratoxins:	OTA	OTA	Pena et al (2006)
Thailand	60	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones:	AFM1 FB1, FB2 OTA DON, DON3GlcA, DON15GlcA, DOM-1, NIV, T-2, HT-2 ZEN, ZEN14GlcA, α-ZEL, β-ZEL	AFM1 OTA DON3GlcA, DON15GlcA	Warth et al (2014)
China	60 adult women	Trichothecenes:	Total DON (free and DONGlcA)	Total DON	Turner et al (2011b)
Spain	56	Aflatoxins: Ochratoxins: Zearalenones:	AFB2, AFG2 OTA, OTB ZEN, α-ZEL	AFB2, AFG2 OTB	Pallarés et al (2022)
Bangladesh	54 pregnant women	Trichothecenes:	Total DON, DOM-1	Total DON	Ali et al (2015)
South Africa	53 adult females	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones:	AFM1 FB1, FB2 OTA DON, DON3GlcA, DON15GlcA, DOM-1, NIV, T-2, HT-2 ZEN, α-ZEL, β-ZEL, ZEN14GlcA	 FB1 OTA DON, DON3GlcA. DON15GlcA, NIV ZEN, α-ZEL, β-ZEL	Shephard et al (2013)

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
Italy	52	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones:	AFM1 FB1 OTA DON, DOM-1 ZEN, α-ZEL, β-ZEL	AFM1 FB1 OTA DON ZEN, α-ZEL, β-ZEL	Solfrizzo et al (2014)
Germany	50 adults	Ochratoxins:	OTA, OTα	OTA, OTα	Ali et al (2017b)
United Kingdom	50	Ochratoxins:	OTA	OTA	Gilbert et al (2001)
Germany	48 adults	*Alternaria* toxins:	TeA, *allo*-TeA	TeA, *allo*-TeA	Hövelmann et al (2016)
Lebanon	44 children	Ochratoxins:	OTA	OTA	Al Ayoubi et al (2021)
Portugal	43 adults	Ochratoxins:	OTA	OTA	Duarte et al (2009)
Tunisia	42 women	Zearalenones:	ZEN, α-ZEL, β-ZEL, α-ZAL, β-ZAL, ZAN	ZEN, α-ZAL, β-ZAL	Belhassen et al (2014)
Sri Lanka	41 (patients with chronic kidney disease and controls)	Aflatoxins: Fumonisins: Ochratoxins:	Total aflatoxins Total fumonisins Total ochratoxins	Total aflatoxins Total fumonisins Total ochratoxins	Desalegn et al (2011)
Croatia	40 pregnant women	Trichothecenes: Ochratoxins:	DON, DON3GlcA, DON15GlcA OTA	DON, DON3GlcA, DON15GlcA OTA	Šarkanj et al (2013)
United Kingdom	35 adults	Trichothecenes:	Total DON (free and DONGlcA)	Total DON	Turner et al (2010b)
United Kingdom	34 adults	Trichothecenes:	DON, DOM-1	DON, DOM-1	Turner et al (2011a)
Amazon	30 adults	Aflatoxins:	AFM1	AFM1	Higashioka et al (2021)

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
Germany	30 adults (17 mill workers)	Ochratoxins: Trichothecenes: Zearalenones: Citrinin:	OTA, OTα DON, DOM-1 ZEN, α-ZEL, β-ZEL CIT, HO-CIT	OTA, OTα DON, DOM-1 ZEN, α-ZEL, β-ZEL CIT, HO-CIT	Föllmann et al (2016)
Belgium	29 adults	Aflatoxins: Fumonisins: Ochratoxins: Trichothecenes: Zearalenones: Citrinin:	AFB1, AFB2, AFG1, AFG2, AFM1 FB1, FB2, FB3 OTA, OTα DON, DON3GlcA, DON15GlcA, DOMGlcA, 3AcDON15GlcA, DOM-1, 15AcDON3GlcA, 15AcDON, 3AcDON, FUSX, DAS, HT-2, T-2 α-ZEL, β-ZEL, α-ZEL7GlcA, α-ZEL14GlcA, β-ZEL14GlcA, ZEN, ZEN14GlcA CIT, HO-CIT	 OTA DON, DON3GlcA, DON15GlcA, DOMGlcA CIT, HO-CIT	Huybrechts et al (2015)
Austria	27 adults	Trichothecenes:	DON, DON3GlcA, DON15GlcA	DON, DON3GlcA, DON15GlcA	Warth et al (2012b)
Iran	17 cancer patients 10 controls	Trichothecenes: Zearalenones:	DON, 3AcDON, 15AcDON, FUSX, DAS, NIV, NEO, T-2, HT-2 ZEN, ZAN	DON, NEO, T-2, HT-2	Niknejad et al (2021)
Spain	23 adults	Ochratoxins: Trichothecenes:	OTA DON, DOM-1, DON3GlcA, 3AcDON, DON3Glc	OTA DON, DOM-1, DON3GlcA, 3AcDON, DON3Glc	Vidal et al (2016)
Malaysia	22 adults	Aflatoxins:	AFM1	AFM1	Sabran et al (2012)
China	15 adult females	Trichothecenes:	DON	DON	Meky et al (2003)
United Kingdom	15 adults	Trichothecenes:	Total DON (free and DONGlcA), DOM-1	Total DON, DOM-1	Gratz et al (2014)
Germany	13 adults	Ochratoxins:	OTA, OTα	OTA, OTα	Muñoz et al (2010)
Chile	12 infants	Ochratoxins:	OTA	OTA	Muñoz et al (2014)

Country	Size	Mycotoxins monitored		Mycotoxins detected	Reference
Spain	10 adults	Aflatoxins:	AFB1, AFB2, AFG1, AFG2		Escrivá et al (2017)
		Ochratoxins:	OTA		
		Zearalenones:	ZEN		
		Enniatins:	**EnA**, **EnA1**, **EnB**, **EnB1**	**EnA1**, **EnB**, **EnB1**	
		Others:	**BEA**		

Compounds most frequently detected in studies of the indoor environment, as per Table 4, are indicated in bold. *Could not chromatographically separate DON3GlcA and DON15GlcA so signal assumed to be sum of both (DONGlcA). ^Reanalysis of samples assessed by Ezekiel et al (2014) using a more sensitive method. 10-OH-OTA, 10-hydroxy-ochratoxin; 15AcDON, 15-acetyldeoxynivalenol; 15AcDON3GlcA, 15-acetyldeoxynivalenol-3-glucuronide; 2'R-OTA, 2'R-ochratoxin A; 3AcDON, 3-acetyldeoxynivalenol; 3AcDON15GlcA, 3-acetyldeoxynivalenol-15-glucuronide; 3'-OH-T-2, 3'-hydroxy T-2 toxin; 3'OH-T-2-3-Glc, 3'-hydroxy T-2 toxin 3-glucoside; 4-deAc-NEO, 4-deacetylated neosolaniol; 4-OH OTA, 4-hydroxy-ochratoxin; 4'OH-T-2-3-Glc, 4'-hydroxy T-2 3-glucoside; AFB1, aflatoxin B1; AFB1-lysine, aflatoxin B1-lysine adduct; AFB1-N7-Gua, 8,9-dihydro-8-(N7-guanyl)-9-hydroxyaflatoxin B1 (DNA adduct); AFB2, aflatoxin B2; AFB3, aflatoxin B3; AFG1, aflatoxin G1; AFG2, aflatoxin G2; AFM1, aflatoxin M1; AFM2, aflatoxin M2; *allo*-TeA, *allo*-tenuazonic acid; ALT, altenuene; ALT1, altertoxin I; AME, alternariol monomethyl ether; AOH, alternariol; BEA, beauvericin; CIT, citrinin; CPA, cyclopiazonic acid; DAS, diacetoxyscirpenol; DOM-1, de-epoxydeoxynivalenol; DOMGlcA, de-epoxy-deoxynivalenol-glucuronide; DON, deoxynivalenol; DON15GlcA, deoxynivalenol 15-glucuronide; DON3Glc, deoxynivalenol 3-glucoside; DON3GlcA, deoxynivalenol 3-glucuronide; DONGlcA, deoxynivalenol glucuronide; Eco, ergocornine; Econ, ergocorninine; Ecr, ergocristine; Ecrn, ergocristinine; Ek, ergokryptine; Ekn, ergokyptinine; Em, ergometrine; Emn, Ergometrinine; EnA, enniatin A; EnA1, enniatin A1; EnB, enniatin B; EnB1, enniatin B1; Es, Ergosine; Et, ergotamine; Etn, Ergotaminine; FB1, fumonisin B1; FB2, fumonisin B2; FB3, fumonisin B3; FUSX, fusarenon-X; HFB1, hydrolysed fumonisin B1; HO-CIT, dihydrocitrinone; HT-2, HT-2 toxin; HT-2-3-GlcA, HT-2 3-glucuronide; HT-2-4-GlcA, HT-2 4-glucuronide; MAS, mono-acetoxyscirpenol; NEO, neosolaniol; NEO3Glc, neosolaniol-3-glucoside; NIV, nivalenol; OH-OTA, hydroxy-ochratoxin; OTA, ochratoxin A; OTB, ochratoxin B; OTα, ochratoxin α; PAT, patulin; ROQC, roquefortine C; STE, sterigmatocystin; T-2, T-2 toxin; T-2-3-GlcA, T-2 toxin 3-glucuronide; TeA, tenuazonic acid; TEN, tentoxin; VER, verrucarol; ZAN, zearalenone; ZAN14GlcA, zearalanone-14-glucuronide; ZEN, zearalenone; ZEN14GlcA, zearalenone-14-glucuronide; ZEN-14-sulfate, zearalenone-14-sulfate; ZENGlu, zearalenone glucoside; α-ZAL, α-zearalanol; α-ZEL, α-zearalenol; α-ZEL14GlcA, α-zearalenol-14-glucuronide; α-ZEL7GlcA, α-zearalenol-7-glucuronide; α-ZELGlcA, α-zearalenol glucuronide; β-ZAL, β-zearalanol; β-ZEL, β-zearalenol; β-ZEL-14GlcA, β-zearalenol-14-glucuronide; β-ZEL-GlcA, β-zearalenol-glucuronide.

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