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Review

Overall internal exposure to mycotoxins and their occurrence in occupational and residential settings – An overview

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ABSTRACT

Mycotoxins are toxic secondary metabolites of various fungal species that can contaminate food and feed, as well as indoor environments. Numerous studies have summarized the adverse health effects of mycotoxins and described severe intoxications of humans and animals. The major health concerns are caused via the alimentary route which unambiguously is the main source for human internal exposure; however, the relevance of other pathways under environmental and occupational conditions should also be considered. Thus firstly, this review aims in summarizing literature data on potentially inhalable mycotoxins occurring in dusts or air in residences and in working environments. Secondly, it gives an overview of the overall internal body burden of mycotoxins in humans in an attempt to characterize total human exposure. These data are also discussed in relation to the current toxicologically based values used for risk assessment.

Contents

Abbreviations: α-ZAL, α-zearalanol; α-ZEL, α-zearalenol; AFB_1, aflatoxin B_1; AFB_2, aflatoxin B_2; AFM_1, aflatoxin M_1; β-ZAL, β-zearalanol; β-ZEL, β-zearalenol; CIT, citrinin; DON, deoxynivalenol; ENN, enniatins; FB_1, fumonisin B_1; FX, fusarenon-X; LOD, limit of detection; LOQ, limit of quantification; M/P, milk-to-plasma ratio; NIV, nivalenol; OTA, ochratoxin A; OTα, ochratoxin α; OTB, ochratoxin B; OTβ, ochratoxin β; PAT, patulin; SG, satratoxin G; SH, satratoxin H; STC, sterigmatocystin; TDI, tolerable daily intake; ZAN, zearalanone; ZEN, zearalenone.

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1. Introduction

Mycotoxins are harmful to human and animal health and have received substantial attention since the discovery of aflatoxins in the 1960s (Asao et al., 1963). Since that time, mycotoxin-producing fungi, their different chemical classes of secondary metabolites and their toxicological properties and associated health concerns have become a challenging issue for researchers worldwide. Individuals can be exposed to mycotoxins via the alimentary or inhalative route, or occasionally via skin or mucosa contact. Today, approximately 400 secondary fungal metabolites are known to be mycotoxins, possessing carcinogenic, cytotoxic, neurotoxic, tremorgenic, immunosuppressive, estrogenic, teratogenic, hepatotoxic, and nephrotoxic effects. Amongst others, *Aspergillus*, *Penicillium*, and *Fusarium* unambiguously belong to the most important genera of mycotoxin-producing fungi in food and feed (Bennett and Klich, 2003). As a consequence of ingestion of contaminated food regulations to limit the exposure of the general population have been in place for decades in approximately 100 countries (Van Egmond et al., 2007). Important mycotoxins regulated in food and feed by legislation of the EU are listed in Table 1.

In the late 1980s, mycotoxins were also recognized as being relevant to environmental and occupational health. Croft et al. (1986) reported one of the first cases of airborne trichothecene toxicosis in a moldy indoor environment. The symptoms resembled stachybotryotoxicosis originally known from horses exposed to moldy straw. Since then, mycotoxins and air contamination have become an integral part of research and are regularly addressed in relevant scientific congresses and journals. The first scientific conference on inhalative mycotoxin exposure and consequences for individuals took place in 1994 in New York, USA (Johanning and Yang, 1995). Afterwards, this conference topic became part of the German Mycotoxin Workshop in 1996 (Gareis and Scheuer, 1996). People living or working in water-damaged buildings, archives, cereal storage facilities, farms, composting plants, as well as modern office buildings equipped with ventilation/air conditioning (HVAC) systems can be exposed to mycotoxins via the inhalative route (Fischer and Dott, 2003; Nielsen, 2003; Jarvis and Miller, 2005). This can be a result of any (unknown) mold growth in the indoor living or working environment (e.g., on wallpapers, gypsum boards, carpets or other bulk materials, as well as in HVAC systems) or of handling mycotoxin-containing food, feed or waste. Depending on the substrates, growth conditions (e.g., temperature, pH, water activity), and properties of the fungal species, the spectrum of molds and mycotoxins that can possibly become airborne is very broad. Indoor exposure usually includes metabolites of *Alternaria*, *Aspergillus*, *Cladosporium*, *Penicillium*, or *Stachybotrys* species (Johanning et al., 1996; Andersson et al., 1997; Nielsen et al., 1998; Richard et al., 1999; Vesper et al., 2000; Skaug et al., 2001a; Gottschalk et al., 2006; Peitzsch et al., 2012). Particularly in sensitized individuals already a small quantity of inhaled spores, fungal fragments, mycotoxins or other microbial metabolites can lead to allergies (e.g., asthma, allergic rhinosinusitis, hypersensitivity pneumonia), and to headache, nausea, fatigue, arthritis and other unspecific symptoms (Redlich et al., 1997; Burge, 2004; Horner et al., 1995). The acute inhalative exposure to *Stachybotrys chartarum* toxins in indoor environments in Cleveland, Ohio, resulted in severe cases of pulmonary hemosiderosis in infants (Dearborn et al., 1999) which can likely be regarded as index cases for the ongoing research and discussion of indoor mold growth and related hazards (Jarvis and Miller, 2005). Nevertheless, CDC concluded that a possible association between acute pulmonary hemorrhage/hemosiderosis in infants and exposure to molds, specifically *Stachybotrys atra*, was not proven (CDC, 2000). Thrasher et al. (2012) described severe health effects in a family occupying a water-damaged building. Here, mycotoxin testing in indoor environments has been connected with human biomonitoring.

Regarding exposure of workers in farms or composting plants, *Fusarium* toxins (which are produced pre-harvest in the field) as well as certain *Aspergillus*, *Penicillium*, and *Stachybotrys* toxins are of importance. They are primarily produced during unsuitable, wet storage conditions and have been shown to occur in respirable dusts (Hintikka, 1978; Sorenson et al., 1984; Fischer et al., 1999; Mayer et al., 2007; Lanier et al., 2012).

In addition to the aforementioned EC-regulated mycotoxins listed in Table 1, important, not regulated fungal metabolites and basic toxic properties are provided in Table 2. Other less frequently examined metabolites that can occur in bioaerosols are the fumigaclavines, trypacidin, and tryptoquivaline produced by *Aspergillus fumigatus* (Fischer et al., 1999), kojic acid produced by *Aspergillus flavus*/*Aspergillus oryzae*, and meleagrin produced by *Penicillium brevicompactum* (Fischer et al., 2000). These compounds will likely receive more attention in the future when being regularly measured (Täubel et al., 2011). There is little information in literature regarding the secondary fungal metabolites of the very frequently occurring *Cladosporium* (C.) spp. Cladosporin, produced from *Cladosporium cladosporioides* and also known as asperentin, acts as a growth inhibitor against fungi; however, its effects on mammals are unknown (Wang et al., 2013). *Alternaria* spp. are commonly found on indoor materials, but little is known about the occurrence of their toxins in bulk samples, air, or dust. Ren et al. (1998) showed that at least two of the *Alternaria* toxins, alternariol and alternariol methyl ether, could be produced on artificially inoculated ceiling tiles. Recently, alternariol, alternariol methyl ether, and altenuene were measured in building materials and indoor dust samples in a study from Finland (Täubel et al., 2011). Additionally, beauvericin, certain enniatins and other less-studied metabolites were demonstrated to be indoor contaminants using multi-analyte methods.

Table 1
Mycotoxins or groups of mycotoxins regulated in food and feed by legislation of the European Commission.

Mycotoxin/group of mycotoxins	Main producing fungal species[a]	Toxic properties[a]	Occurrence	Commission legislation
Aflatoxins B1/B2, G1/G2, M1	*Aspergillus* (*A.*) *flavus*, *A. parasiticus*	Hepatotoxic, carcinogenic, immuno-suppressive	Nuts, seeds, dried fruits, cereals, maize, milk (Aflatoxin M1 after carry-over)	Food: Regulation (EC) No 1881/2006; Feed (Aflatoxin B1): Directive 2002/32/EC
Citrinin	*Monascus ruber; Penicillium* (*P.*) *citrinum; Aspergillus* spp.	Nephrotoxic, genotoxic, carcinogenic	Fermented rice products	Food: Regulation (EC) No 1881/2006
Deoxynivalenol (type B-trichothecene)	*Fusarium* (*F.*) *graminearum*, *F. culmorum*	Cytotoxic, gastrointestinal symptoms, immuno-suppressive	Cereals and maize and products thereof	Food: Regulation (EC) No 1881/2006; Feed: Recommendation 2006/576/EC
Fumonisins B_1/B_2	*F. verticillioides*, *F. proliferatum*; *A. niger*[b]	Carcinogenic, edematous, membrane damages	Maize and maize products	Food: Regulation (EC) No 1881/2006; Feed: Recommendation 2006/576/EC
Ochratoxin A	*A. ochraceus*, *A. niger*; *P. chrysogenum*	Carcinogenic, nephrotoxic, teratogenic, immunotoxic	Cereals, raisins, coffee, wine, spices, liquorice	Food: Regulation (EC) No 1881/2006; Feed: Recommendation 2006/576/EC
Patulin	*Aspergillus* spp.; *Penicillium* spp.; *Byssochlamys* spp.	Genotoxic, neurotoxic	Fruits and juices, particularly apples	Food: Regulation (EC) No 1881/2006
T-2/HT-2 toxin (type A-trichothecene)	*F. sporotrichioides*, *F. langsethiae*[c]	Cytotoxic, gastrointestinal symptoms, immuno-suppressive	Cereals, particularly oats	Under evaluation
Zearalenone	*F. graminearum, F. equiseti, F. culmorum, F. verticillioides*	Estrogenic, reproductive toxicity	Cereals and maize and products thereof, maize oil	Food: Regulation (EC) No 1881/2006; Feed: Recommendation 2006/576/EC
Rye ergot *sclerotia*	*Claviceps* (*C.*) *purpurea, C. fusiformis*	Ergot alkaloids[d] contained in sclerotia: gangrenous, neurotoxic	Cereals	Feed: Directive 2002/32/EC (amount of sclerotia, not of ergot alkaloids)

[a] Compiled from Cole et al. (2003), SCF (1999–2002), Bennett and Klich (2003), Richard (2007); findings based on animal models, in vitro-studies or epidemiological data.
[b] Frisvad et al. (2007).
[c] Torp and Nirenberg (2004).
[d] Ergot alkaloids (e.g. ergovalin) can also be produced by the endophytic fungi *Neotyphodium* or *Epichloe* on grasses (Richard, 2007).

Table 2
Moulds and mycotoxins of importance in indoor environments and for occupational health (in addition to the EC-regulated compounds listed in Table 1).

Mycotoxin/group of mycotoxins[a]	Main producing fungal species[a]	Toxic properties[a]
Alternaria toxins[b]	*Alternaria* spp., *Alternaria alternata*	Genotoxic, teratogenic, carcinogenic
Atranones A-K	*Stachybotrys* (*S.*) *chlorohalonata*, *S. chartarum* chemotype A	Proinflammatory
Beauvericin[c]	*Fusarium* spp., *Beauveria bassiana*	Cytotoxic, immunotoxic
Chaetoglobosines, chaetomin, chaetoviridins	*Chaetomium globosum, Chaetomium* spp.	Cytotoxic
Enniatins[c]	*Fusarium* spp.	Cytotoxic, immunotoxic
Fumitremorgens	*Aspergillus* (*A.*) *fumigatus*	tremorgenic
Gliotoxin	*Aspergillus* spp., *A. fumigatus*, *Penicillium* (*P.*) spp.	Cytotoxic, immunosuppressive
Mycophenolic acid	*Penicillium brevicompactum, P. roquefortii, Penicillium* spp.	Immunosuppressive
Penitrem A	*Penicillium* spp.	Tremorgenic
Roquefortin C	*P. roquefortii, P. chrysogenum*	Neurotoxic
Roridins (E, L-2, and epi-/isomers)[d]	*S. chartarum* chemotype S	Cytotoxic, immunosuppressive
Satratoxins (G, H, F and isomers)[d]	*S. chartarum* chemotype S	Cytotoxic, immunosuppressive
Spirocyclic drimanes	*S. chlorohalonata*, *S. chartarum* chemotype A and S	Immunosuppressive, anti-inflammatory, cytotoxic, neurotoxic
Sterigmatocystin, 5-methoxy-sterigmatocystin	*A. versicolor*	Carcinogenic
Trichodermol, Trichodermin	*S. chlorohalonata*, *S. chartarum* chemotype A, *Memnoniella echinata*	Cytotoxic
Verrucarin J[d]	*S. chartarum* chemotype S	Cytotoxic, immunosuppressive
Verrucologen	*A. fumigatus, Penicillium* spp.	Tremorgenic

[a] Compiled from Betina (1989), Andersen et al. (2003), Cole et al. (2003), Nielsen et al. (1999), Nielsen (2003), Jarvis and Miller (2005), Rand et al. (2006), Pestka et al. (2008), Täubel et al. (2011), EFSA (2014); findings based on animal models, in vitro-studies or epidemiological data.
[b] Alternariol, alternariol methyl ether (AME), altenuene, tenuazonic acid, tentoxin, altertoxins (ATX I-III), AAL-toxins (EFSA, 2014). To date, proof of production on indoor samples only for alternariol and AME (Ren et al., 1998) and altenuene (Täubel et al., 2011; Peitzsch et al., 2012). Only limited information on their toxic potential to humans.
[c] Newly discovered as indoor contaminants (Täubel et al., 2011; Peitzsch et al., 2012).
[d] Usually occurring together (Andersen et al., 2003).

Currently, such LC–MS/MS-based approaches are the methods of choice due to their sensitivity and selectivity, as well as their ease of detection of multiple toxins in a single run (Gottschalk et al., 2006; Delmulle et al., 2006; Bloom et al., 2007; Vishwanath et al., 2009; Täubel et al., 2011). Another important fungus is *Chaetomium* (*Ch.*) of which certain species like *Chaetomium globosum* are able to produce Chaetglobosins and azophilones (McMullin et al., 2013). Furthermore, there are still species newly detected in indoor

environments which are able to produce different, less studied secondary metabolites, i.e. *Aspergillus insuetus* and *Aspergillus calidoustus* or various *Eurotium* spp. (Slack et al., 2009). Also *Wallemia*, commonly known as spoilage organism of salted food, was recently described to occur in indoor environments (Desroches et al., 2014). Other indoor molds and their secondary metabolites were extensively reviewed by Miller and McMullin (2014).

Regarding mycotoxins in the environment, some countries have recommendations for mold spores/m^3 air, but not for airborne mycotoxins (WHO, 2009). However, it is not elucidated if spores are the major way of mycotoxin release into the air. Brasel et al. (2005) have shown that mycotoxins also occur on particulates smaller than fungal spores. This is supported by findings of Gareis and Gareis (2007) and Gareis and Gottschalk (2014), which showed that exudates segregated by some molds contain high concentrations of mycotoxins. Furthermore it was shown that mycotoxins can be absorbed and distributed by mites (*Tyrophagus casei*). This led to the assumption that their feces regarded as important house dust allergens possibly also contain mycotoxins (Gareis and Göbel, 1998). It is unclear whether the toxicity of mycotoxins inhaled in aerosolized form or bound to spores or dusts differs in comparison with intake via food or feed. Kelman et al. (2004) even discussed whether the concentrations of inhalable mycotoxins in indoor environments could reach levels sufficient to present a risk to individuals. However, workers with airway exposure to *Aspergillus flavus*-contaminated dust had an elevated risk of hepatocellular carcinoma compared to those without exposure (Lai et al., 2014).

Few animal studies have focused on the inhalative toxicity of aerosolized T-2 toxin. Actually, this toxin is not found indoors but is an important representative of trichothecenes and *Fusarium* toxins. Thus, it may have an impact on occupational health of farm workers. Their results, however, are conflicting and influenced by the way and duration of administration (Robbins et al., 2000). Creasia et al. (1990) reported an up to 20-fold higher inhalative toxicity in a study with rats and guinea pigs compared to an intraperitoneal exposure. Similar results were obtained with mice (Creasia et al., 1987). However, in a porcine animal model, the animals were approximately seven-fold less susceptible to airborne T-2 toxin than via intravenous administration (Pang et al., 1988). Another study with guinea pigs indicated the subcutaneous administration of T-2 toxin to be approximately twice toxic as inhalative exposure (Marrs et al., 1986).

On the one hand, this review aims to provide a comprehensive compilation of literature data on the occurrence of potentially inhalable mycotoxins in dusts or air in residences and workplaces. The occurrence of molds and mycotoxins in indoor materials such as wallpapers, gypsum boards, or ceiling tiles is well documented in literature but was not in the scope of this review due to a missing direct linkage to airborne mycotoxin exposure. Overall, it is very difficult to conduct a risk assessment of airborne mycotoxins due to a lack of reliable data on inhalative toxicity and respirable concentrations in the air, as well as a lack of standardized methods for sampling and examination. This is further aggravated by the individual sensitivity of different people toward (microbial) volatile organic compounds and air contaminants and the multifactural nature of human health disorders in the context of dampness, mold or respiratory dust exposure (WHO, 2009; Burge, 2004). Spores, fungal fragments or microdust particles, mycotoxins, bacteria and actinomycetes, endotoxins (lipospolysaccharides), and (1→3)-β-D-glucans or a combination of them can be responsible for the beforementioned symptoms (Douwes, 2005; Górny et al., 2002; Kuhn and Ghannoum, 2003; Lorenz et al., 2013; Täubel et al., 2011; Reponen et al., 2007; Yike et al., 2007). However, unequivocally, mycotoxins are hazardous compounds and the inhalative exposure to especially highly cytotoxic or carcinogenic compounds should be reduced as far as possible. Other hazardous compounds present in (indoor) air, which unambiguously can be involved in the etiology of the above-mentioned health symptoms as well, were not addressed here.

On the other hand we also discuss current knowledge regarding the overall internal exposure to mycotoxins (i.e., human biomonitoring) and characterize human exposure. These data will be discussed in relation to the toxicological values that are currently used for risk assessment. For this purpose, we summarized the results of different PubMed inquiries to obtain an overview of the current scientific literature. We also included papers presented at conferences, reports from governmental, scientific and other institutions, and, where possible, unpublished reports and other gray literature.

2. External inhalative exposure

2.1. Occurrence at workplaces

The first descriptions of the deleterious health effects of aflatoxins in farm animals appeared in the 1960s. Since that time, increasing evidence has indicated that mycotoxins are a serious health threat in occupational conditions (Bennett and Klich, 2003; Degen, 2011). To verify exposure of workers, a variety of investigations measured the concentrations of mycotoxins (primarily aflatoxins and ochratoxin A) in the air or dust of agricultural environments (see Tables 3 and 4). Most of these studies focused on farms, grain and corn processing facilities, as well as peanut or malt factories.

Large indoor air variations were found for AFB_1 and OTA (0.04–4849 ng/m^3 and 0.00007–14 ng/m^3, respectively). The highest AFB_1 concentrations were observed during bin cleaning activities in 11 farms with a maximum value of 4849 ng/m^3, followed by animal feeding in 9 farms with up to 421 ng/m^3 in the US (Selim et al., 1998). The highest OTA level (14 ng/m^3) was reported during grain emptying in Norwegian farms (Halstensen et al., 2004). High DON levels up to 720 ng/m^3 were found during grain storage and in a cattle farm, whereas ZEN concentrations were low during grain storage (Mayer et al., 2007). Lanier et al. (2010) were the first reporting gliotoxin in bioaerosols during the handling of feed rations of corn silage, oilseed cakes and hay.

The results for mycotoxins in dust samples are presented in Table 4. The highest levels were obtained in settled grain dust collected in 1999–2000 in Norway for HT-2 and T-2 toxin with maximum values of 2400 and 1200 ng/g, respectively (Nordby et al., 2004). Additionally, high concentrations of OTA and citrinin were found during grain storage (Tangni and Pussemier, 2006).

2.2. Occurrence in residences

Limited results were available for residences (Table 5). These results were primarily from water-damaged buildings in Belgium and Germany (Polizzi et al., 2009; Gottschalk et al., 2008). Polizzi et al. (2009) analyzed 20 mycotoxins in 62 of 99 air samples from 7 water-damaged buildings with visible fungal growth and primarily found roquefortine C (maximum 4 ng/m^3), chaetoglobosin A and sterigmatocystin but also roridin E, OTA, AFB_1, and AFB_2. In general, the fungi identified matched well with the mycotoxins detected. In Germany, satratoxin G and H were observed in the airborne dust of a water-damaged dwelling with an observed contamination of *Stachybotrys chartarum* (Gottschalk et al., 2008).

The results of mycotoxins in residential dust are presented in Table 6. Overall, in most of the samples, the concentrations in residences are near or below the LOQ. No clear differences between water-damaged buildings and control buildings without water

Table 3
Concentrations of mycotoxins in the air of workplaces.

References	*N*	*N* > LOQ	Results (ng/m³) median (range)	Remarks
Aflatoxin B_1				
Van Nieuwenhuize et al. (1973)			0.9–72	Peanut mill
Burg et al. (1981)	19	18	0.23–100 AFB	Grain harvesting
Sorenson et al. (1984)	12	10	0.4–7.6	Peanut shelling plant
Selim et al. (1998)	24		0.04–92	Grain harvesting; US; 1989–1993
Selim et al. (1998)	22		5–421	During animal feeding; US; 1989–1993
Selim et al. (1998)	14		124–4849	Bin cleaning; US; 1989–1993
Lanier et al. (2012)	12[c]	0	AFB_1 and AFB_2 <LOQ	1 cattle farm; France
DON				
Lappalainen et al. (1996)	15	2	3 and 20	8 farms; Finland 1993–1994
Mayer et al. (2007)	35		2.0[a] (0.2–720)	Grain storage; Germany; 2005/06
Ochratoxin A				
Mayer et al. (2007)	35		0.002[a] (0.00007–0.69)	Grain storage; Germany; 2005/06
Halstensen et al. (2004)	31		0.04 (0.003–14)	Grain emptying; Norway; 1999–2000
Halstensen et al. (2004)	34		0.04 (0.002–0.6)	Grain storage; Norway; 1999–2000
Iavicoli et al. (2002)	6	6	0.006–0.09	Industries where coffee, cacao and spices were processed; Italy
Gerbl-Rieger et al. (1999)[b]			0.003–0.03	Waste treatment
Zearalenon				
Mayer et al. (2007)	35		1.0 [a] (0.1–50.1)	Grain storage; Germany; 2005/06
Gliotoxin				
Lanier et al. (2010)	3		1100–3700	1 cattle farm; France

[a] Mean.
[b] Cited by Mayer et al. (2008).
[c] 12 days in a stable.

Table 4
Concentrations of mycotoxins in settled dust of workplaces.

References	*N*	*N* > LOQ	Results (ng/g) median (range)	Remarks
Aflatoxin B1				
Van Nieuwenhuize et al. (1973)			–(250–410)	Peanut mill
Selim et al. (1998)	2	2	125, 227[b]	During animal feeding; US; 1989
DON				
Nordby et al. (2004)	104	45	<20 (<20–340)	Farms; Norway; 1999–2000
Ochratoxin A				
Halstensen et al. (2004)	99		4.0 (2–128)	Grain storage; Norway; 1999–2000
Tangni and Pussemier (2006)	4	4	104[a] (17–318)	Grain storage; Belgium; 2001–2003
Skaug et al. (2001c)	14	6	27.5[a] (0.2–70)	Cowshed; Norway
Gareis and Meussdoerffer (2000)	31		0.99[a] (0.05–9.9)	Malt factories; Germany; 1997
Citrinin				
Tangni and Pussemier (2006)	4	4	244[a] (137–344)	Grain storage; Belgium; 2001–2003
HT-2 toxin				
Nordby et al. (2004)	104	92	54 (<30–2400)	Farms; Norway; 1999–2000
T-2 toxin				
Nordby et al. (2004)	104	24	<50 (<50–1200)	Farms; Norway; 1999–2000

[a] Mean.
[b] Single values.

damage or visible mold infestation were observed. Only Charpin-Kadouch et al. (2006) reported significant differences between the trichothecene concentrations in 15 flooded dwellings contaminated by *S. chartarum* or *Chaetomium* and 9 control buildings using an ELISA assay. In a study of 200 dust samples (Ertl et al., 2007) and a more recent preliminary study of 8 samples (Mosch et al., 2015) in Germany (all from buildings without contamination), most of the results for AFB, SG, SH, STC, OTA, and CIT were below the LOQ. Only DON could be quantified in 9% of the samples, with levels between 176 and 8100 ng/g (Ertl et al., 2007).

3. Occurrence in breast milk and exposure of infants

Some data on mycotoxins in breast milk are available in the scientific literature. However, the research has primarily focused on OTA. The results of OTA in breast milk are shown in Table 7. First, Gareis et al. (1988) described detectable levels in 83% of 36 randomly collected samples in Germany (ranging from <10 to 30 ng/l). Subsequently, other studies in Europe showed mean contamination levels between 6.0 ng/l and 30 ng/l. A large variation with very high concentrations was found in only two studies performed in Hungary and Italy in the 1990s (Kovács et al., 1995; Micco et al., 1995). In the first study, 92 colostrum samples were collected in an area with a known high exposure to OTA and a high incidence of an OTA-related disease called porcine nephropathy. In Italy, the authors provided no explanations for the high concentrations, but diet of mothers was not correlated with the observed OTA levels in breast milk. However, other studies showed that breakfast cereals, bakery products, processed meat products, and cheese could be important contributors to dietary OTA intake resulting in higher concentrations of OTA in breast milk (Galvano et al., 2008; Skaug et al., 2001b). Three studies in South America found similar or

Table 5
Concentrations of mycotoxins in indoor air of residences.

References	*N*	*N* > LOQ	Results (ng/m^3) ranges	Remarks
Aflatoxin B_1				
Polizzi et al. (2009)	99	5	0.002–0.15	Belgium; 7 water-damaged buildings
	99	4	0.0003–0.021 AFB_2	
Satratoxins				
Gottschalk et al. (2008)	1		0.25 SG SH	Germany; 1 water-damaged building
Sterigmatocystin				
Polizzi et al. (2009)	99	3	0.003–1.77	Belgium; 7 water-damaged buildings
Roquefortin C				
Polizzi et al. (2009)	99	1	0.009–4.0	Belgium; 7 water-damaged buildings
Roridin E				
Polizzi et al. (2009)	99	1	0.003–0.082	Belgium; 7 water-damaged buildings
Macrocyclic trichothecenes				
Charpin-Kadouch et al. (2006)	15		0.62 ppb[a]	France; moldy dwellings; 2003
	9		0.29 ppb[a]	France; control dwellings; 2003
Ochratoxin A				
Polizzi et al. (2009)	99	1	0.012–0.228	Belgium; 7 water-damaged buildings
Chaetoglobosine A				
Polizzi et al. (2009)	99	3	0.007–3.42	Belgium; 7 water-damaged buildings

[a] ELISA for quantitative detection of trichothecenes, including roridin A, E, H, and L-2, satrotoxin G and H, isosatratoxin F, verrucarin A and I, and verrucarol.

only slightly higher levels but clearly higher concentrations were reported in studies performed in Africa and the Asian region of Turkey. For example, Gürbay et al. (2010) found levels ranging from 621 to 13,111 ng/l in a more recent study from Ankara, Turkey, in which 75 samples were collected in 2007–2008. The highest levels were reported by studies performed in Egypt and Sierra Leone with means of 21,060 ng/l and 7900 ng/l, respectively (AbdAlla et al., 2002; Jonsyn et al., 1995). Only two studies described the ratio of OTA from the mother's plasma to the fetus and to breast milk. In the first study, umbilical cord blood samples and milk samples collected 3–4 days after the delivery of 130 Italian and non-Italian women showed an average milk-to-plasma ratio (m/p) of 0.02 when only values above the LOQ were considered (Biasucci et al., 2011). In a second study, maternal blood and breast milk were collected for up to 6 months in Chile with a higher mean M/P value in colostrum (M/P 0.4) than in mature milk (M/P between 0.09 and 0.26; average 0.25) (Muñoz et al., 2014).

The results for AFM_1 in breast milk were available in only some countries, and primarily prior to 2003 (Table 8). Two studies from Italy and one from the Asian region of Turkey reported low mean concentrations of only <0.5 ng/l, <5.0 ng/l (the LOD), and 0.74 ng/l, respectively (Turconi et al., 2004; Galvano et al., 2008; Atasever et al., 2014). In contrast, high mean levels of 800 ng/l, 560 ng/l, 300 ng/l, and 13.5 ng/l were observed in Sierra Leone, the United Arab Emirates, and Egypt (two studies), respectively (Jonsyn et al., 1995; Abdulrazzaq et al., 2003; AbdAlla et al., 2002; Polychronaki et al., 2006). In the 1990s, samples from Thailand showed higher medians (664 ng/l) compared to samples from Australia (71 ng/l) collected in 1991–1992 (El-Nezami et al., 1995).

Other mycotoxins were measured in only two studies. These studies used LC–MS/MS or UHPLC-HRMS methods with higher LOQs. In a Spanish study conducted in 2012, ZEN could not be detected; however, ZEA was found in 13 of 21 samples with concentrations ranging from 2.1 to 14.3 μg/l (Rubert et al., 2014). HT-2 and NIV were detected in 10 (12.2–62.5 μg/l) and 3 (53.1–69.7 μg/l) of 21 samples. In another study, ZEN, NIV, HT-2, FB1, FX, and DON could not be quantified above the corresponding LOQ's of 0.2, 0.25, 0.25, 0.5, 0.25, and 0.25 μg/l in three breast milk samples collected in 2011 (Cao et al., 2013).

For the majority of mycotoxins, a quantification of the daily intake of an exclusively breastfed infant is not reasonable due to the lack of sufficient exposure data but for OTA the daily intake (DI) could be calculated using the following equation:

$$DI = C_{milk} \times V_{milk} \times R$$

where C_{milk} is the concentration of OTA in breast milk, V_{milk} is the amount of milk ingested daily by an exclusively breastfed infant, and R is the resorption factor. We also used the following assumptions: 4-month-old child, 5 kg body weight, daily breast milk consumption of 159 ml/kg bodyweight (SCF, 2003), and 100% resorption of the target substances from the gastrointestinal tract. In Europe, the mean/median OTA values reported in more recent studies were between 5 and 30 ng/l. This corresponds to a daily intake between 0.8 and 4.8 ng/kg b.w., which is clearly below the TDI value of 17 ng/kg b.w. Additionally, the maximum value of 100 ng/l observed in Germany did not exceed the TDI. Nevertheless, higher concentrations with large variations were described before in Table 7 for other countries. Specifically, high percentages of breastfed infants derived concentrations above the TDI in Turkey, Egypt, and Sierra Leone. In a study performed in Chile, the highest mean OTA intake was found to be 12.7 ng/kg b.w. for newborns; infants at later stages of breastfeeding received only 5 ng/kg b.w. (Muñoz et al., 2014).

4. Human biomonitoring studies in the general population

Substantial data are available from investigations measuring a variety of mycotoxins, especially ochratoxin, trichothecenes, fumonisins, aflatoxins, and zearalenone in human samples across the globe (summarized in Zinedine et al., 2007; Coronel et al., 2010; Leong et al., 2012; Malir et al., 2012; Turner et al., 2012). Various techniques, such as enzyme-linked immunosorbent assay (ELISA), radioimmunoassay (RIA), thin layer chromatography (TLC), gas chromatography–mass spectrometry (GC–MS), liquid chromatography with fluorescence or mass spectrometric detection (HPLC-FLD, HPLC-MS/MS) with various extraction, cleanup, and enrichment methods, have been applied (Capriotti et al., 2012; Nesic et al., 2014). More recently, multibiomarker studies were performed to characterize the concentrations of various mycotoxins in exposed persons, as well as in the general population (Warth et al., 2013a).

Table 6
Concentrations of mycotoxins in settled dust of residences.

References	*N*	*N* > LOQ	Results (ng/g) ranges	Remarks
Aflatoxin				
Ertl et al. (2007)	200	0	<8.6	Germany; control buildings
Mosch et al. (2015)	8		<1.3	
Beauvericin				
Täubel et al. (2011)	4	4	0.05–1.4	Finland; 9 water-damaged residences and 2 public buildings
Satratoxins				
Bloom et al. (2009b)	7	0	n.d. SG n.d. SH	USA; 5 water-damaged homes
Bloom et al. (2009a)	18	0	n.d. SG n.d. SH	Sweden; 8 water-damaged buildings
Mosch et al. (2015)	8		<7 SH <13 SG	
Sterigmatocystin				
Bloom et al. (2007)	8	1	17	Sweden; 4 water-damaged buildings
Bloom et al. (2009a)	18	0	<LOD	Sweden; 8 water-damaged buildings
Bloom et al. (2009b)	7	2	16 and 28	USA; 5 water-damaged homes
Engelhart et al. (2002)	11	2	2.0 and 4.0	Germany; 8 moldy or water-damaged buildings
Ertl et al. (2007)	200	4	<3.2 to >108	Germany; control buildings
Mosch et al. (2015)	8	0	<0.7	Germany; control buildings
Verrucarol[a]				
Bloom et al. (2009a)	18	2	0.6 and 1.7	Sweden; 8 water-damaged buildings
Bloom et al. (2007)	8	2	19 and 43	Sweden; 4 water-damaged buildings
Trichodermol				
Bloom et al. (2007)	8	2	2.4 and 3.4	Sweden; 4 water-damaged buildings
Bloom et al. (2009b)	7	0	n.d.	USA; 5 water-damaged homes
DON				
Ertl et al. (2007)	200	18	<176–8100	Germany; control buildings
Mosch et al. (2015)	8	0	<13	Germany; control buildings
Gliotoxin				
Ertl et al. (2007)	200	1	<15–110	Germany; control buildings
Mosch et al. (2015)	8	0	<13	Germany; control buildings
Trichothecenes				
Charpin-Kadouch et al. (2006)	15		21.9 ppb	France; moldy dwellings; 2003
	9		0.31 ppb	France; control dwellings; 2003
Ochratoxin A				
Kasel et al. (1999)	47	13	<0.5–5.1	Germany; 47 control buildings
Richard et al. (1999)	1	0	<0.5	USA
Töpfer et al. (2010)	50	5	<0.23–23.9	Germany; control buildings
	43	8	<0.23–14.6	Germany; moldy buildings
Ertl et al. (2007)	200	0	<43	Germany; control buildings
Mosch et al. (2015)	8	0	<3	Germany; control buildings
Chaetoglobosine				
Täubel et al. (2011)	4	1	3100	Finland; 9 water-damaged residences and 2 public buildings
Emodin				
Täubel et al. (2011)	4	4	0.24–84	Finland; 9 water-damaged residences and 2 public buildings
Enniatin B				
Täubel et al. (2011)	4	4	0.63–11	Finland; 9 water-damaged residences and 2 public buildings
Citrinin				
Ertl et al. (2007)	200	0	<8.6	Germany; control buildings

[a] Hydrolysis product of macrocyclic trichothecenes (Harrach et al., 1981).

4.1. Aflatoxins

Aflatoxins, especially the most toxic and frequently occurring AFB_1, were summarized in several reviews (Jager et al., 2011; Leong et al., 2012; Turner et al., 2012). After ingestion, AFB_1 is rapidly absorbed and primarily metabolized to the highly reactive 8,9-epoxide, which binds covalently to DNA and serum albumin, thereby producing AFB_1-N^7-guanine and lysine adducts (AF-Alb). AFB_1 can additionally be oxidized to several other metabolites, especially AFM_1. AFM_1 and AFB_1-N^7-guanine in urine, as well as AF-Alb adducts in serum, are reliable biomarkers, and the latter represents a reliable marker of long-term intake. Some studies reported a significant correlation between AF-Alb and AFM_1 levels (Gan et al., 1988; Piekkola et al., 2012).

Studies reporting on AFM_1 levels in urine are summarized in Table 9. Overall, the concentrations in Europe are currently under

Table 7
Concentrations of OTA in breast milk.

References	*N*	*N* > LOQ	Results (ng/l) (medians and ranges)	Remarks
Europe				
Gareis et al. (1988)	36	4	–(<10–30)	Germany; 1986
Breitholtz-Emanuelsson et al. (1993)	40	23	–(<10–40)	Sweden; 1990/91
Zimmerli and Dick (1995)	40	4	<5.0 (<5–14)	Switzerland; 1992/3
Kovács et al. (1995)	92[b]	38	<200 (<200–7630)	Hungary; 1992
Micco et al. (1995)	111	22	<100 (<100–12,000)	Italy
Miraglia et al. (1995)	3[c]	3	<60 (<60–540)	Italy
Skaug et al. (1998)	115	38	<10 (<10–130)	Norway; 1994
Skaug et al. (2001b)	80	17	<10 (<10–182)	Norway; 1995/6
Postupolski et al. (2006)	13	5	<5.0 (<5.0–17)	Poland; 1998/99
Turconi et al. (2004)	231	167	4.0 (<0.5–57)	Italy; 1999
Galvano et al. (2008)	82	61	30.4[a] (<5–405)	Italy; 2006
Dostal et al. (2008)	76	23	<4.8 (<4.8–60.3)	Slovakia; 2007
Biasucci et al. (2011)	129	41	10.0[a] (<0.5–75.1)	Italy; 2007
Muñoz et al. (2013)	30	7	24.4[a] (<10–100)	North Rhine-Westfalia; 2009
	60	6	14.4[a] (<10–78)	Lower Saxonia; 2006–2010
Africa, Asia, America				
Jonsyn et al. (1995)	113	40	7900[a] (200–337000)	Sierra Leone
AbdAlla et al. (2002)	120	43	21,060 (5070–45010)	Egypt; 2000–2002
Navas et al. (2005)	50	2	<10 (10 and 20)	Brazil; 2001/2
Hassan et al. (2006)	50	36	1890[a] (–)	Egypt
Gürbay et al. (2010)	75	75	–(621–13,111)	Turkey; 2007/8
Muñoz et al. (2010b)	9[b]	9	106[a] (44–184)	Chile; 2008/9
Muñoz et al. (2014)	21	17	52[a] (<10–186)	Chile; 2008–2010
Dehghan et al. (2014)	87	84	24.6[a] (<LOD–60.0)	Iran; 2011
Andrade et al. (2013)	224	0	<10	Brazil; 2011–2012

[a] Mean.
[b] Colostrums.
[c] Over some consecutive days.

Table 8
Concentrations of AFM_1 in breast milk.

References	*N*	*N* > LOQ	Results (ng/l) (medians and ranges)	Remarks
Europe				
Turconi et al. (2004)	231	1	<0.5 (194)	Italy; 1999
Galvano et al. (2008)	82	4	<7.0 (<7.0–140)	Italy; 2006
Africa, Asia, America				
Zarba et al. (1992)	5	5	–(?–1.4)	The Gambia
Jonsyn et al. (1995)	113	35	800[a] (200–99000)	Sierra Leone
El-Nezami et al. (1995)	7311	11 5	71 (28–1038) 664 (39–1736)	Australia; 1992 Thailand; 1991
Abdulrazzaq et al. (2003)	140	129	560 (?–3400)	United Arab Emirates; 1999–2000
Navas et al. (2005)	50	1	<10 (20)	Brazil; 2001/2
AbdAlla et al. (2002)	120	66	300 (200–2090)	Egypt; 2000–2002
Polychronaki et al. (2006)	388	138	4.2 (<4.2–5000)	Egypt; 2003
Ghiasian and Maghsood (2012)	132	8	10.0 (<5.0–10.8)	Iran; 2003–2004
Keskin et al. (2009)	61	8	<5.0 (<5.0–6.9)	Turkey; 2006–2007
Elzupir et al. (2012)	94	51	0.21 (<0.01–2.56)	Sudan
Atasever et al. (2014)	73	18	0.74[a] (<LOD–6.0)	Turkey; 2008–2009
Adejumo et al. (2013)	50	41	–(<10–92.1)	Nigeria; 2010
Cao et al. (2013)	3	1	70	China; 2011
Andrade et al. (2013)	224	0	<10	Brazil; 2011–2012
Omar (2012)	80	80	67.8[a] (9.7–137.2)	Jordan; 2011–2012

[a] Mean.

or slightly above the LOQ. For example, Gerding et al. (2014) could not quantify AFM_1 above 0.005 μg/l in 101 samples from adults in Germany. In Asia, several studies in recent decades showed primarily low levels, whereas distinctly higher levels were observed for some individuals. In a large cross-sectional study, urine was collected from 600 volunteers in China, Zhejiang province from 2010 to 2012 (Lei et al., 2013). AFM_1 was detected in 85% of the samples with levels up to 4.9 μg/l and with a higher percentage of higher values in persons from rural areas compared to those from urban regions. The scenario in Africa is also variable. Low levels were reported for South Africa, Egypt, and Guinea (Shephard et al., 2013; Hatem et al., 2005; Polychronaki et al., 2008), higher values were found in Nigeria, Cameroon, and Gambia (Ezekiel et al., 2014; Abia et al., 2013; Jonsyn-Ellis, 2007). The highest levels ever found were reported in a study carried out in Sierra Leone in 1992–1993 (Jonsyn-Ellis, 2000). Here, 244 primary school children showed levels up to 374 μg/l.

AF-Alb has been analyzed in a variety of studies, notably in African countries, such as Ghana (Obuseh et al., 2010; Shuaib et al., 2010; Tang et al., 2009), Gambia (Wild et al., 1992, 1993, 2000; Turner et al., 2007; Castelino et al., 2014, 2015), Guinea (Gong et al., 2002), Egypt (Piekkola et al., 2012; Turner et al., 2008c), Tanzania (Shirima et al., 2013; Routledge et al., 2014), and Kenya (Yard et al., 2013), as well as in Asian countries such as China (Wang et al., 1996; Cheng et al., 1997; Sylla et al., 1999; Chen et al., 2013), Taiwan (Cheng et al., 1997; Ahsan et al., 2001), Nepal (Groopman et al.,

Table 9
AFM_1 in the urine of the general population.

References	*N*	*N* > LOQ	Results (μg/l) median (range)	Remarks
Europe				
Malir et al. (2006)	205	118	0.127 (0.019–19.2) pg/mg crea	Czech Republic; 1997/98
Solfrizzo et al. (2014)	52	3	0.07 (<0.02–0.15)	Italy; 2011
Huybrechts et al. (2014)	32	0	<0.005	Belgium
Njumbe Ediage et al. (2012)	40	0	<0.02	Belgium
Gerding et al. (2014)	101	0	<0.005	Germany; 20–30 years
Asia/America				
Zhu et al. (1987)	42		0.42[a] (0.01–3.2) ppb	China; 1985
Qian et al. (1994)	317	67	–(<0.04–5.2)	China; 1986–1989
Cheng et al. (1997)	138	88	3.2 (?–108) ng/12 h (China)	China and Taiwan; 1989
	32	21	2.7 (?–17) ng/12 h (Taiwan)	
Sun et al. (1999)	145	78	0.01 (0.003–0.243)	China; 1987–1998
Mykkänen et al. (2005)	300	142	0.04 (0.004–0.561)	China
Ahn et al. (2010)	12	1	0.002	Korea; adults and one child
Redzwan et al. (2012)	160	98	0.023[a] (<0.011–0.075)	Malaysia; 23–57 years
Lei et al. (2013)	600	504	0.06 (<0.1–4.9)	China; 2010–2012
Warth et al. (2014)	60	3	–(<0.15–0.55)	Thailand; 10–76 years
Romero et al. (2010)	69	54	0.006[a] (<0.002–0.04)	Brazil; 2005
Johnson et al. (2010)	179	21	141.5 (1.89–935) pg/mg crea	USA; 2007–2008
Africa				
Nyathi et al. (1987)	1228	1007	4.2[a]	Zimbabwe; 1984/85
Jonsyn-Ellis (2000)	234	104	–(0.5–374) (dry season)	Sierra Leone; 1992/93; children
	190	97	–(0.1–124) (rainy season)	
Hatem et al. (2005)	60	6	–(0.01–0.07)	Egypt; 6–20 months
Jolly et al. (2006)	91	83	473 (<LOD–11,562) pg/mg crea	Ghana; 2002
Jonsyn-Ellis (2007)	131	39	0.7[a] (<LOQ–6.8)	Gambia; 5–14 years
Polychronaki et al. (2008)	50	4	0.003[b] (<0.005–0.006)	Egypt; 2003; 1,1–2,5 years
	50	32	0.016[b] (<0.005–0.801)	Guinea; 2003; 2–4 years
Piekkola et al. (2012)	93	44	5.48[b] (<4.1–409) pg/mg crea	Egypt; 2006; 18–40 years
Abia et al. (2013)	145	15	0.05[a] (<0.05–1.38)	Cameroon; 2011
Shephard et al. (2013)	53	0	<0.01	South Africa; 19–97 years
Njumbe Ediage et al. (2013)	220	31	0.33[a] (<0.01–4.7)	Cameroon; 1.5–4.5 years
Kouadio et al. (2014)	99	40	–(<0.06–14.1)	Ivory Coast: 2011; 2–70 years
Ezekiel et al. (2014)	120	17	0.3[a] (<0.15–1.5)	Nigeria; 2012

[a] Mean.
[b] Geometric mean.

2014), India (Anitha et al., 2014), and Bangladesh (Groopman et al., 2014). Very high AF-Alb concentrations with medians/averages from 11 to 110 pg/mg albumin and maximum values up to approximately 1000 pg/mg were found in regions of the aforementioned countries. In these particular regions endemic primary liver cancer and high levels of food products contaminated with aflatoxins were observed. Nevertheless, substantial AF-Alb concentration variability was found between geographic regions within a single country (Wild et al., 2000; Yard et al., 2013; Castelino et al., 2015; Routledge et al., 2014; Groopman et al., 2014). Conversely, in developed countries, such as France and Poland, AF-Alb was not detected above the LOQ (Wild et al., 1990). In the southwest US, AF-Alb was detected in 21% of 184 blood samples with measureable concentrations between 1.0 and 16.6 pg/mg albumin (Wild et al., 2000; Johnson et al., 2010; Castelino et al., 2015; Routledge et al., 2014; Groopman et al., 2014). Several studies indicate contaminated food to be the primary source of AF-Alb, and the consumption of maize-based products is especially associated with high levels of AF-Alb (Gan et al., 1988; Sylla et al., 1999; Johnson et al., 2010; Shirima et al., 2013; Chen et al., 2013; Routledge et al., 2014). Only two studies measured the concentrations of AF-Alb in cord blood and maternal blood. In Gambia, median levels were 10.1 pg/mg albumin in cord blood and 40.4 pg/mg albumin in maternal blood with a good correlation between both sample series (Turner et al., 2007). In the second, performed in Bangladesh, 18.1 and 25.4 pg/mg albumin levels were observed in first and third trimester samples, whereas 27.4 pg/mg albumin was observed in cord blood (Groopman et al., 2014). The effects of a changing food policy in response to diminishing maize consumption were investigated in China (Chen et al., 2013). The median AF-Alb levels declined from 19.3 pg/mg albumin in 1989 (100% above the LOQ) to <0.5 pg/mg albumin in 2012 (7% above the LOQ). A summary of various intervention trials for reducing AFB exposure was compiled by Kensler et al. (2011).

4.2. Ochratoxins

OTA was rapidly absorbed from the gastrointestinal tract of monogastric animals with differences in the percentage of OTA absorbed between species (Pfohl-Leszkowicz and Manderville, 2007; Han et al., 2013; Vettorazzi et al., 2014). Elimination half-lives in rodents and pigs after oral administration depending on dose and are slow (1.6–10 days) (Bauer and Gareis, 1987; Hagelberg et al., 1989; Zepnik et al., 2003; Kuiper-Goodman et al., 2010). In humans and non-human primates, OTA binds strongly to blood proteins and was excreted with a plasma half-life of 35.5 days in humans, predominantly as OTA and its metabolite OTα via the kidneys (Hagelberg et al., 1989; Studer-Rohr et al., 2000; Wu et al., 2011; Haighton et al., 2012). Therefore, OTA levels in serum and plasma are suitable biomarkers of exposure. Levels in urine are also useful; however, these concentrations are considerably lower (Muñoz et al., 2010a). Ochratoxin B (OTB), another secondary metabolite with lower toxicity compared to OTA, is excreted after a single oral dose as 10% OTB and 3% OTβ in the urine of rats and additionally 6% was excreted in feces (Mally et al., 2005).

The results of OTA in blood from the general population with no known exposure are given in Table 10. Overall, the blood concentrations were low in Europe with medians/means ranging from 0.13 to 1.1 μg/l. Some researchers found higher individual levels up to 19.4 μg/l in the Czech Republic, Bulgaria and Croatia, primarily in studies performed in the 1990s. Significantly higher concentrations

Table 10
Concentrations of OTA in blood of the general population.

References	*N*	*N* > LOQ	Results (μg/l) median (range)	Remarks
Europe				
Breitholtz et al. (1991)	297	38	–(<0.3–6.7)	Sweden; 1989
Ruprich and Ostry (1993)	594	240	0.20 (>0.05–37.0)	Czech Republic; 1990–1991; 18–58 years
Breitholtz-Emanuelsson et al. (1994)	65		0.44[a] (0.12–2.0)	Italy; 1992
Zimmerli and Dick (1995)	205 (m)	205	0.30 (0.06–6.02)	Switzerland; 1992/93; 20–40 years
	163 (f)	163	0.24 (0.10–1.84)	
Märtlbauer et al. (2009)	102	100	0.33 (0.07–1.29)	Germany; 1990–1997
Palli et al. (1999)	138	134	0.48 (0.12–2.84)	Italy; 1994; 35–65 years
Solti et al. (1997)	355	290	–(<0.2–10.0)	Hungary; 1995
Peraica et al. (1999)	249	147	0.39[a] (0.2–15.9)	Croatia; 1997
Thuvander et al. (2001)	200		0.17 (0.01–0.48)	Sweden; 1997
Thuvander et al. (2001)	206		0.16 (0.05–0.42)	Norway; 1998
Rosner et al. (2000)	927	909	0.23 (<0.06–2.03)	Germany; adults and chidren; various cities
Degen et al. (2003)	84		0.39 (?–2.0)	EU; different countries
Petkova-Bocharova et al. (2003)	17	17	1.6[a] (0.1–10.9)	Bulgaria
Postupolski et al. (2006)	30	30	1.0 (0.14–3.41)	Poland; 1998/99
Skaug (2003)	104		0.42 (0.036–5.534)	Norway; 2000; adults
Ostry et al. (2005)	2206	2077	0.2 (<0.1–13.7)	Czech Republic; 1994–2002
Grajewski et al. (2007)	6	6	0.37[a] (0.32–0.4)	Poland; 2005; 40–68 years
Muñoz et al. (2010a)	13	13	0.24 (0.19–0.29)	Germany; 2008
		13	1.14 (0.07–1.64) ($OT\alpha_{Total}$)	
Medina et al. (2010)	168	168	1.09 (0.15–5.71)	Spain; 2008; adults
Coronel et al. (2009)	279	275	0.54 (0.11–8.68)	Spain; 18 to >45 years
Gilbert et al. (2001)	50	50	–(0.15–2.17)	UK
Di Giuseppe et al. (2012)	327	3234	0.17 (<0.03–2.92)	Italy; 38–48 years
Dohnal et al. (2013)	115	115	0.14 (0.037–1.13)	Czech Republic; 19–40 years
Malir et al. (2013)	100	96	0.13 (<0.10–0.35)	Czech Republic; females; 19–40 years
Cramer et al. (2015)	50[b]	50	0.21 (0.07–0.38)	Germany; 18 to >60 years
Asia				
Ueno et al. (1998)	184	156	0.07[a] (0.004–0.28)	Japan; 1992–1996
Özcelik et al. (2001)	40		0.44[a] (0.19–1.43)	Turkey
Aslam et al. (2005)	31	30	0.31[a] (0.04–1.24)	Pakistan
Africa				
Maaroufi et al. (1995a)	140	73	–(0.1–8.8)	Tunesia
Maaroufi et al. (1995b)	17		–(0.1–2.3)	Tunesia; 1992–1993
Abid et al. (2003)	185		3.4[a] (1.2–5.5)	Tunesia; 1991–2000
Assaf et al. (2004)	250	83	0.17[a] (0.1–0.87)	Lebanon; 2001–2002
Filali et al. (2002)	309	185	0.29[a] (0.08–6.59)	Marocco; 2000; 18–60 years
Sangare-Tigori et al. (2006)	63	22	0.29[a] (0.01–5.81)	Ivory Coast; 2001–2004
Jonsyn-Ellis (2007)	131	25	2.4[a] (<LOQ–60)	Gambia; 5–14 years
Grosso et al. (2003)	62		0.53[a] (0.16–8.06)	Tunesia
Hassen et al. (2004)	40	28	2.6[a] (<LOQ–7.5)	Tunesia
Hmaissia Khlifa et al. (2008)	105	29	0.49[a] (0.12–3.4)	Tunesia
Zaied et al. (2011)	138	68	3.3[a] (1.7–8.5)	Tunesia; 2007–2009
Ibrahim et al. (2013)	22		0.2[a] (0.005–0.50)	Egypt; 2010–2012
Americas				
Scott et al. (1998)	144		0.81 (0.29–2.37)	Canada; 1994
Muñoz et al. (2006)	54	22	0.44 (0.07–2.75)	Chile; 2004
	44	40	0.77 (0.22–2.12)	
Pacin et al. (2008)	199	125	0.11/0.24 (<0.019–74.8)	Argentina; 2004–2005; two regions
Guzman et al. (2007)	149	142	0.62[a] (0.01–1.9)	Costa Rica

f: females; m: males.
[a] Mean.
[b] Dried blood spots.

were found in some North African countries, such as Tunisia, as well as in a study from Argentina.

Results in cord blood are limited. OTA was found in 99% of 130 Italian cord blood samples with levels ranging from 0.08 to 4.84 μg/L (mean: 0.5 μg/l) (Biasucci et al., 2011). In 70 German samples, the levels ranged from <0.06 to 0.90 μg/l (median: 0.37 μg/l) (Rosner et al., 2000).

Some studies investigated the factors that influence the OTA body burden; however, the results are largely inconclusive. The consumption of several foods, including cereal products, wine, beer, fruit juice, chocolate, and coffee, were weakly related to high plasma levels of ochratoxin A (Thuvander et al., 2001; Klapec et al., 2012; Coronel et al., 2009; Medina et al., 2010; Biasucci et al., 2011; Di Giuseppe et al., 2012; Malir et al., 2013). In a study collecting duplicate diet samples and corresponding urine samples of 50 volunteers, a significant correlation was reported (Gilbert et al., 2001). Table 11 summarizes the concentrations of OTA in urine. Overall, the levels in Europe and Asia are low with medians/means between 0.01 and 0.14 μg/l; higher values were more frequent in some African countries.

Since the 1950s, a chronic disease referred to as Balkan endemic nephropathy (BEN) has been observed with high incidences in some regions of Romania, Bulgaria and the former Yugoslavia (Pfohl-Leszkowicz et al., 2002). Epidemiologic studies have

Table 11
Concentrations of OTA in urine of the general population.

References	*N*	*N* > LOQ	Results (μg/l) median (range)	Remarks
Europe				
Pena et al. (2006)	60	42	0.038[a] (0.021–0.105)	Portugal; 2004; 19–82 years
Duarte et al. (2010)	155	122	0.018[a] (<0.008–0.07)	Portugal; 2007; 18–96 years
Muñoz et al. (2010a)	13	13	0.05 (0.02–0.14))OTA)	Germany; 2008
		13	2.09 (0.49–7.12) ($OT\alpha_{Total}$)	
Solfrizzo et al. (2011)	10	10	0.05 (0.02–0.25)	Italy; 26–87 years
Gilbert et al. (2001)	50	46	–(<0.01–0.058)	UK
Rubert et al. (2011)	27	3	–(<0.5 to <1.5)	Spain; 2010; 21–77 years
Wallin et al. (2015)	252	128	0.046[a]	Sweden; 2010/11
Solfrizzo et al. (2014)	52	52	0.14 (?–2.13)	Italy; 2011
Šarkanj et al. (2013)	40	4	<0.17 (<0.05 to <0.17)	Croatia; 2011; 26–33 years
Klapec et al. (2012)	40	23	0.02 (<0.016–1.11)	Croatia; females; 36–33 years
	40	40	1.18 (0.11–7.57) (OTα)	
Njumbe Ediage et al. (2012)	40	4	–(<0.06–0.61)	Belgium; adults
		3	–(<0.06–15) ($OT\alpha_{Total}$)	
Huybrechts et al. (2014)	32	22	0.01[a] (<0.03–0.033)	Belgium
Gerding et al. (2014)	101	0	<0.025	Germany
Africa				
Jonsyn-Ellis (2000)	234	64	–(0.07–148)	Sierra Leone; 1992/93
	190	47	–(0.6–72.2)	
Abia et al. (2013)	145	25	0.08[a] (<0.17–1.87)	Cameroon; 2011; 18–58 years
Njumbe Ediage et al. (2013)	220	70	0.2[b] (0.04–2.4)	Cameroon; 1.5–4.5 years
Kouadio et al. (2014)	99	37	–(<0.01–0.42)	Ivory Coast; 2011; 2–70 years
Shephard et al. (2013)	54	52	0.024 (<0.002–0.432)	South Africa; 19–97 years
Ezekiel et al. (2014)	120	34	0.2 (<0.15–0.6)	Nigeria; 2012
Asia				
Ahn et al. (2010)	12	12	0.031[a] (0.013–0.093)	Korea; adults and one child
Warth et al. (2014)	60	1		Thailand; 10–76 years
Xie et al. (2014)	65	5	–(<LOQ–0.091)	China
America				
Brewer et al. (2013)	104	87	6.2[a] (2.0–14.6 ppb)	USA; 2012; adults

f: females; m: males.
[a] Mean.
[b] Geometric mean.

indicated that mycotoxins play an etiologic role because the populations were exposed to high levels of OTA in food and because very high levels of OTA were found in the blood of the affected populations. OTA has also been found more frequently in the urine of people living in BEN-endemic villages than in those in non-endemic villages; the highest levels were observed in patients with BEN or urinary tract tumors (Radic et al., 1997; Petkova-Bocharova et al., 2003; Castegnaro et al., 2006). Some studies from Tunisia suggested that OTA blood levels are higher in people with chronic interstitial nephropathy than in healthy individuals (Maaroufi et al., 1995a,b; Abid et al., 2003; Hmaissia Khlifa et al., 2008; Hassen et al., 2004; Zaied et al., 2011). Additionally, there have been indications that patients suffering from other kidney diseases have higher blood levels (Breitholtz-Emanuelsson et al., 1994; Özcelik et al., 2001; Aslam et al., 2005; Grajewski et al., 2007). Nevertheless, the weight of evidence for a causal relationship between OTA and BEN was critically discussed (Mally et al., 2007). For example, Bui-Klimke and Wu (2015) summarized in a review that all epidemiologic studies showed no statistically significant evidence for human health risks associated with OTA exposure or have methodological limitations.

4.3. Trichothecenes

4.3.1. Deoxynivalenol

DON is efficiently absorbed from the gastrointestinal tract of monogastric animals and humans and are rapidly excreted in the urine, primarily in a glucuronidated form (Thieu and Pettersson, 2009; Dänicke and Brezina, 2013; Warth et al., 2013b).

In humans, approximately 86–90% of DON was excreted as DON-15-β-D-O-glucuronide (D15GlucA) and, to a lesser extent, as DON-3-β-O-glucuronide (D3GlucA) (Turner et al., 2010a; Warth et al., 2012a, 2013b). Total DON was frequently measured in urine samples after enzymatic hydrolysis via treatment with β-glucuronidase. The results of studies quantifying DON in urine are summarized in Table 12. Mean total DON concentrations in European countries ranged from 2.9 to 48.7 μg/l. The highest levels were reported in Croatia and Austria, and the lowest levels in Sweden and Italy. The mean values are also low in various African and Asian countries with the exception of China (20.4 μg/l). Nevertheless, all of these studies showed very high concentrations for some individuals, particularly in rural parts of Croatia, with peak levels up to 1238 μg/l. Some studies found no relationship between urine DON excretion and food intake (Gerding et al., 2014); others observed a significant relationship with grain/cereal intake (Meky et al., 2003; Turner et al., 2008a, 2010b; Wallin et al., 2013) or maize intake (Turner et al., 2010b; Srey et al., 2014; Shephard et al., 2013). Additionally, one study showed the efficiency of a wheat restriction intervention diet to reduce DON levels (Turner et al., 2010a). In a recent study, Gratz et al. (2014) reported annual variations of DON exposure quantified from urine levels.

4.3.2. Nivalenol and fusarenon-X

Only very few data from toxicokinetic studies of nivalenol (NIV) and 4-acetylnivalenol (fusarenon-X, FX) were available from animals but not for humans. In pigs, only 17% of the administered NIV dose was excreted in the urine without any further metabolism; however, the NIV in the feces was primarily unchanged (Hedman et al., 1997). In mice, ^{3}H-NIV was given p.o., and radioactivity was primarily excreted via the feces (Poapolathep et al., 2003). In contrast, FX was more rapidly and more efficiently absorbed from the

Table 12
Concentrations of DON, FB1 and ZEN in the urine of the general population (total DON if not otherwise stated).

References	*N*	*N* > LOQ	Results (μg/l) median (range)	Remarks
DON				
Turner et al. (2011)	60	58	4.8[a] (<0.5–29.9)	China; 1997/1998; 40–70 years
Turner et al. (2010a)	35	35	11.6[a] (0.5–78.2)	UK; 21–59 years (normal diet)
Turner et al. (2010b)	76	75	6.8 (<0.5–28.8)	France; 1997–2000 (farmers)
Turner et al. (2008b)	25	25	10.8 (0.7–61.3)	UK
Turner et al. (2008a)	300	296	8.9[b] (0.6–48.2) ng/mg crea	UK, 2000–2001; 19–64 years
Turner (2010)	29	28	10.8 (<LOD–65.8)	Sweden
Piekkola et al. (2012)	93	63	1.1[a] (<0.5–59.9) ng/mg crea	Egypt; 2006
Lattanzio et al. (2011)	2	2	3 and 8 free DON	Italy; 2006–2007
Wallin et al. (2013)	326	292	2.9 (<0.5–65.8)	Sweden; 2009–2011; 18–80 years
Solfrizzo et al. (2011)	10	7	3.7[a] (<0.8–14.2)	Italy; 26–87 years
Cunha and Fernandes (2012)	13		16.3[a] (<0.5–26.2)	Portugal; 20–50 years
Šarkanj et al. (2013)	40	31	48.7 (4.8–1238)	Croatia; 2011
Solfrizzo et al. (2014)	52	50	11.9[a] (<1.5–67.4)	Italy; 2011
Warth et al. (2012b)	27	26	20.4(<LOD–63)	Austria
Njumbe Ediage et al. (2012)	40	5	–(<0.7–68.3) free-DON	Belgium; adults
Njumbe Ediage et al. (2013)	220		2.2[b] (0.1–77)	Cameroon; 1.5–4.5 years
Abia et al. (2013)	145	62	5.9[a] (<10–74.7)	Cameroon; 2011
Shephard et al. (2013)	53	46	3.1 (<0.5–53.4)	South Africa; 19–97 years
Meky et al. (2003)	15	15	12[a] (4–18)	China (low risk area)
Srey et al. (2014)	166		2.5[b]	Tanzania; 2011; 6–14 months
Kouadio et al. (2014)	99	21	–(<0.8–10.0)	Ivory Coast; 2011; 2–70 years
Ezekiel et al. (2014)	120	6	3.9[a] (<4.0–10.0)	Nigeria; 2012
Huybrechts et al. (2014)	32	32	82[a] (3.0–420) DON-15GlcA	Belgium
Warth et al. (2014)	60	7	8.1[a] (<3.0–16.8) DON-15GlcA	Thailand; 10–76 years
Gerding et al. (2014)	101	84	9.4 (3.0–139) ng/mg crea DON-GlcA	Germany; 20–30 years
Rodríguez-Carrasco et al. (2014)	54	37	–(1.6–30.4) free-DON	Spain; 2013: 8- > 28 years
Fumonisin B_1				
Gong et al. (2008)	24	11	0.03[b] (0.02–0.065[c])	Mexico
Xu et al. (2010)	43	36	3.91[a] (0.06–253.61) ng/mg crea	China; 2005; 25–65 years; two
	34	28	0.39[a] (0.01–3.72) ng/mg crea	different regions
Ahn et al. (2010)	12	0	<0.02	Korea
Van der Westhuizen et al. (2011)	44	43	0.25[b] (0.14–0.35[c])	South Africa; 19–97 years
Wallin et al. (2015)	252	15	0.004[a]	Sweden; 2010/11
Abia et al. (2013)	145	5	0.63[a] (<1.7–14.8)	Cameroon; 2011
Shephard et al. (2013)	53	49	0.4 (<0.12–4.9)	South Africa
Warth et al. (2012b)	27	0	<0.17	Austria
Njumbe Ediage et al. (2012)	40	0	<0.1	Belgium
Riley et al. (2012)	177	101	0.3	Guatemala; 2011; 17–76 years
Shirima et al. (2013)	147	141	(<0.02–0.44)	Tanzania; 2011, 18–22 months
Njumbe Ediage et al. (2013)	220	24	2.96[b] (<0.03–48)	Cameroon; 1.5–4.5 years
Torres et al. (2014)	1240		(<0.1–46.6)	Guatemala; 2011/12; 18–70 years
Solfrizzo et al. (2014)	52	29	0.06 (<0.01–0.35)	Italy, 2011; 26–87 years
Huybrechts et al. (2014)	32	0	<0.2	Belgium
Ezekiel et al. (2014)	120	16	4.6 (<2.0–12.8)	Nigeria; 2012
Warth et al. (2014)	60	0	<0.2	Thailand; 10–76 years
Gerding et al. (2014)	101	0	<0.01	Germany; 20–30 years
Zearalenone (ZEN)				
Warth et al. (2012b)	27	0	<0.13	Austria
Abia et al. (2013)	145	4	0.22[a] (<LOQ–1.42) (free-ZEN)	Cameroon; 2011
	145	7	0.74[a] (<LOQ–21.4) (total-ZEN)	
Njumbe Ediage et al. (2012)	40	4	–(<1.2–12.6)	Belgium; adults
Njumbe Ediage et al. (2013)	220	9	0.97 b (0.03–5.0)	Cameroon; 1.5–4.5 years
Wallin et al. (2015)	252	92	0.03[a]	Sweden; 2010/11
Solfrizzo et al. (2014)	52	52	0.06 (0.01–0.12)	Italy; 2011; 26–87 years
Ezekiel et al. (2014)	120	1	0.3[a] (free-ZEN)	Nigeria; 2012; 1–80 years
		8	9.5[a] (?–44.5) (total-ZEN)	
Shephard et al. (2013)	53	53	0.20 (0.08–3.15)	South Africa; 19–97 years
Gerding et al. (2014)	101	0	<0.005	Germany; 20–30 years
Bandera et al. (2011)	163	90	0.38 (<0.05–33.1)	USA; 9–10 years
Warth et al. (2014)	60	0	<0.6	Thailand; 10–76 years
Huybrechts et al. (2014)	32	0	<0.05	Belgium

[a] Mean.
[b] Geometric mean.
[c] 95% confidence limits.

gastrointestinal tract of mice than NIV and was further metabolized to NIV in the liver and kidney (Poapolathep et al., 2003). Again, Saengtienchai et al. (2014) reported that FX has a high oral bioavailability and is rapidly converted to NIV in piglets and excreted into urine with an elimination half-life of 1.7 h. Therefore, the concentration of NIV in urine samples reflects not only the intake of NIV but also that of FX. Warth et al. (2012b, 2014) were not able to quantify NIV above the LOQs of 1.0 and 4.0 μg/l in Austria and Thailand, respectively, whereas in South Africa and Cameroon, levels from <3 to 3.7 μg/l and <10 to 22.0 μg/l, respectively, were observed (Shephard et al., 2013; Abia et al., 2013).

4.3.3. T-2 and HT-2 toxin

Information on the toxicokinetics of T-2 and HT-2 toxins is incomplete. T-2 toxin is rapidly metabolized to a large number of products with HT-2 toxin and T-2 triol as the primary metabolites (excreted via the kidney) (Yagen and Bialer, 1993; EFSA, 2011b; Sun et al., 2014). In mice and guinea pigs orally or i.m. administered ^{3}H-labeled T-2, 62% and 57% of the dose were recovered in the urine, respectively (Matsumoto et al., 1978; Pace et al., 1985). In two experiments performed by Robison et al. (1979), 22% and 18% were recovered in urine after the oral administration of ^{3}H-labeled T-2 toxin to pigs. No information on the toxicokinetics for humans is currently available. In one case of a human suicide attempt up to 3.2 ng/ml HT-2 toxin and 21.2 ng/ml T-2 tetraol were quantified in urine after swallowing 20–25 mg T-2 toxin and 5 mg T-2 tetraol. In a stomach lavage sample of the subject also no T-2 toxin was detectable but HT-2 toxin and T-2 tetraol (SCOOP, 2003).

T-2 toxin and HT-2 toxin were not found to be above the LOQ (Rubert et al., 2011; Njumbe Ediage et al., 2012; Warth et al., 2012b, 2014; Gerding et al., 2014; Huybrechts et al., 2014; Rodríguez-Carrasco et al., 2014).

4.3.4. Macrocyclic trichothecenes

The cumulative excretion of satratoxin G (SG) in feces and urine was monitored over 5 days following intranasal instillation in mice (Amuzie et al., 2010). Total fecal and urinary excretion accounted for only a small fraction (0.3%) of the total dose. The authors speculated that SG was metabolized by phase I and/or phase II enzymes yielding products that could not be quantified via the ELISA used for analysis. Trichothecene mycotoxins in sera from individuals exposed to indoor molds and controls were analyzed using a competitive ELISA that was highly specific for macrocyclic trichothecenes (Brasel et al., 2004). The majority of the samples show concentrations below or slightly above the limit of detection (0.1 μg/l). However, more results above the LOD (78%) were observed in the group with known exposure. For the clinical diagnosis of exposure, a satratoxin G albumin adduct assay was performed by Yike et al. (2006). Serum samples from three patients with documented exposure to *S. chartarum* similarly revealed lysine, cysteine, and histidine adducts. These adducts were also found in the sera of rats exposed to the spores of *S. chartarum* but not in control human subjects or animals.

4.3.5. Verrucarol

Verrucarol is a trichothecene that is structurally related to T-2 and HT-2 toxins. It is not a naturally occurring compound but formed after hydrolysis of macrocyclic trichothecenes from *S. chartarum* (Harrach et al., 1981). Following oral administration in dogs, the absolute bioavailability ranged from 14% to 102% (mean: 44%) with a terminal half-life of 1.6 h (Barel et al., 1990). Analysis of urine showed that <1% of the administered i.v. dose was excreted unchanged with urine. Metabolites were not reported and no other toxicokinetic data or results from biomonitoring studies of the general population are currently available.

4.4. Fumonisins

Various fumonisin analogs have been described, and the fumonisin B series predominated exposure results (Turner et al., 2012). Fumonisin B types were poorly absorbed after oral dosing in animals and were primarily excreted unchanged in feces. Fumonisin B types did not undergo significant metabolism and were rapidly excreted with urine, primarily as FB_1 (Riley et al., 2012) (relevant study results are summarized in Table 12). FB_1 concentrations were close or below its LOQ in all studies from Europe and one in Thailand; however, higher levels were reported in some African countries, as well as in a region from China and for high maize consumers in Guatemala (Gong et al., 2008). Additional studies suggested that maize consumption contributes to fumonisin exposure (Gong et al., 2008; Torres et al., 2014; Van der Westerhuizen et al., 2013; Shirima et al., 2013); however, the levels also depend on the agroecological characteristics of the region (Torres et al., 2014).

4.5. Zearalenone

The metabolism of zearalenone (ZEN) in humans is not thoroughly understood; however, animal data have suggested that ZEN is metabolized primarily to alpha-zearalenol (α-ZEL) and beta-zearalenol (β-ZEL) with further conversion to alpha-zearalanol (α-ZAL) and beta-zearalanol (β-ZAL), respectively (Kleinova et al., 2002; Zinedine et al., 2007). α-ZAL is further metabolized into β-ZAL and, to a lesser extent, into zearalanone (ZAN). In pigs and humans, glucuronidated ZEN and α-ZEL were the primary metabolites detected in urine (Mirocha et al., 1981; Farnworth and Trenholm, 1983; Olsen et al., 1985; Döll et al., 2003; Thieu and Pettersson, 2009; Brezina et al., 2014). Bandera et al. (2011) quantified the metabolites and found that ZEN was predominant, followed by β-ZEL and β-ZAL in a study of New Jersey (USA) with female children. In all other studies only some quantifiable amounts near the LOQ were observed (see Table 12). Nevertheless, studies in Belgium and the US reported maximum ZEN concentrations of up to 12.6 μg/l and 33.1 μg/l, respectively. In two studies, the total ZEN was quantified as a sum of the various metabolites and glucuronidation products. A mean value of 0.74 μg/l (0.22 μg/l ZEN) was observed in Cameroon (Abia et al., 2013); 9.5 μg/l (0.3 μg/l ZEN) was observed in Nigeria (Ezekiel et al., 2014).

4.6. Citrinin

Toxicokinetic studies describing the oral pathway are scarce. In rats, citrinin is eliminated predominantly via the kidney, as described in a study with radiolabelled citrinin by Reddy et al. (1982) in which approximately 75% of the subcutaneous dose was recovered in urine.

In another study, rats were dosed orally with ^{14}C-CIT. A total of 74% of the radioactivity appeared in the urine in the first 24 h (Phillips et al., 1979). Additionally, ^{14}C-labeled citrinin was given orally to rats, and unchanged citrinin and dihydrocitrinone (DH-CIT) were responsible for 43% and 15% of the total radioactivity detected in urine, respectively (Dunn et al., 1983).

In the first biomonitoring study, CIT was present in all plasma samples from 8 German adults at concentrations ranging from 0.11 to 0.26 μg/l (Blaszkewicz et al., 2013). CIT was also detected in 8/10 urine samples from 4 other adults and 6 infants at a range of 0.16–0.79 μg/l. The same study group reported median CIT and DH-CIT (the main metabolite) levels of 0.03 μg/l (<0.02–0.08 μg/l) and 0.06 μg/l (<0.05–0.51 μg/l) in the urine of 50 adults in Germany (Ali et al., 2014a). Moreover, CIT and DH-CIT were detectable in 94% and 71% of the urine samples from rural (n = 32) and urban (n = 37) areas of Bangladesh with median levels of 0.08 μg/l and 0.03 μg/l, respectively, for CIT and 0.2 μg/l and 0.05 μg/l, respectively, for DH-CIT (Ali et al., 2014b). Two other researchers could not quantify CIT above an LOQ of 5.7 μg/l in Cameroon or above 0.03 μg/l in Belgium (Njumbe Ediage et al., 2012; Huybrechts et al., 2014).

4.7. Patulin

Toxicokinetic studies involving PAT are very limited. Up to 29% of PAT was rapidly absorbed within one hour after the application

of high doses in a perfused rat model (Rychlik et al., 2005). In rats, 39% of the orally administered ^{14}C-labeled PAT was eliminated with urine, primarily within 48 h calculated according to the measured radioactivity (Dailey et al., 1977). PAT quickly reacts with glutathione in erythrocytes to form a complex mixture of adducts (Fliege and Metzler, 2000). In a preliminary survey, PAT was analyzed in the serum of 5 volunteers drinking apple juice. There were no measureable amounts above the detection limit of 0.2 μg/l (Rychlik, 2003). Additionally, in three experiments, a volunteer drank 1 L of apple juice that was adjusted to a PAT content of approximately 50 μg/l (Rychlik, 2005). Again, no PAT was detected in serum samples prior to or after consumption.

4.8. Sterigmatocystin

In rats, peak plasma levels were reached after 30 min following an oral bolus administration of ^{14}C-STC and were eliminated in the feces (64–92%) and urine (approximately 5–15% depending on maturity) (Walkow et al., 1985). The absorption of STC was also studied in a monkey given a single oral dose of ^{14}C-labeled STC (Steyn and Thiel, 1976). The maximal rate of absorption from the gastrointestinal system was not higher than 30–50% of the radioactivity recovered in the urine as glucuronic acid conjugates of sterigmatocystin. No information regarding humans was available, and there was no data from biomonitoring studies.

4.9. Enniatins

Enniatins are *Fusarium* toxins representing a large group of approximately 29 naturally occurring analogs of which four (enniatins A, A1, B, and B1) have been most frequently detected in foods and feeds (EFSA, 2014). No TDI value was recommended by the EFSA because there is a substantial lack of toxicological data. In a preliminary study, after feeding rats with an ENN-rich feed, ENN could be detected in several organs and in the blood (Manyes et al., 2014). Urine specimens were not analyzed in this experiment. An in vitro study using Caco-2 cells supported a high bioavailability of ENN with absorption values ranging from 57% to 70% (Meca et al., 2012). In a pilot toxicokinetic study with five pigs, ENN B1 was rapidly absorbed after oral administration with an oral bioavailability of 91% (Devreese et al., 2014). Subsequently, the mycotoxin is rapidly eliminated with a terminal half-life of 1.6 h. No data on the concentrations excreted into urine were given in this study.

ENN B concentrations in human urine samples of 101 Germans were reported by Gerding et al. (2014). ENN B could be detected in 20% of the samples, but all values were below the LOQ of 0.0013 μg/l.

5. Human biomonitoring studies in occupational settings

5.1. Aflatoxins

Autrup et al. (1991) analyzed AFB_1 serum albumin adducts among Danish animal feed production workers. Seven out of 45 workers were positive. Viegas et al. reported occupational exposure in 31 workers involved in poultry production with levels ranging from <1.0 to 4.23 μg/l but not in controls (Viegas et al., 2012). In a second study, AFB_1 was detected in 41 workers from a waste company and 30 controls (Viegas et al., 2014). All workers showed levels from 2.5 μg/l to 25.9 μg/l, whereas all of the controls had values below the LOQ. Considerably higher levels (a mean of 189 μg/l AFB_1) were observed in feed mill workers in India (Oluwafemi et al., 2012). Bronchoalveolar lavage (BAL) and serum samples from 46 food grain workers and 44 non-food grain workers were analyzed for aflatoxins in India (Malik et al., 2014). AFB and a higher prevalence of respiratory symptoms and *Aspergillus* in BAL culture were detected in 33% of the food grain workers and 9% of the controls.

5.2. Ochratoxin A

In Germany, serum samples from 7 malt factory workers showed OTA levels between 0.13 and 2.6 μg/l (Gareis and Meussdoerffer, 2000). The levels were higher than that of the local population, and the authors concluded that the handling of barley post-harvest and an increased exposure to dust could be responsible for these findings. In 6 Italian workers from industries where coffee, cocoa beans, and spices were processed, serum concentrations ranged from 0.94 to 3.28 μg/l, largely exceeding those of the control group (Iavicoli et al., 2002). OTA levels in blood samples from farm workers and non-farm working controls were examined by Skaug (2003). All serum samples contained OTA (mean 0.4 μg/l); however, the levels in the serum were unrelated to farm working. These results were supported by a German study in which blood samples from 61 male workers employed at granaries were analyzed (Degen et al., 2007). With mean values of 0.28 ng/ml these results gave no indication that the body burden of OTA was influenced by inhalation; however, measurable OTA concentrations have been found in dust samples collected at the corresponding workplaces. Additionally, OTA was analyzed in blood samples from workers employed at waste handling facilities in Germany (Degen et al., 2003). Median serum levels in subgroups of workers involved in waste deposition or in waste sorting were 0.36 and 0.53 μg/l, respectively, indicating an additional uptake of OTA via inhalation in workers with exposure to bioaerosols.

5.3. Deoxynivalenol

In a pilot study of 76 French farmers, the total DON was analyzed in 75 urine samples with levels ranging from 0.5 to 28.8 μg/l (Turner et al., 2010b).

6. Calculation of total intake from biomonitoring data

6.1. Aflatoxin B_1

Some studies have reported on metabolism and toxicokinetics in various species. In rats orally administered ^{3}H-AFB_1, approximately 15% of the radioactivity was observed in the urine (Coulombe and Sharma, 1985); dosing with ^{14}C-AFB_1 resulted in a recovery of 8.8% in the urine (Helferich et al., 1986). Wong and Hsieh (1980) reported that 19% and 25% of a single oral dose was found in rats and mice, respectively, whereas monkeys excreted 38% of the dose. Four pigs were fed diets with three different levels of AFB_1, and urine sampling showed that 22% of the AFB_1 was converted to AFM_1 and excreted (Thieu and Pettersson, 2009). In a Chinese study, two adult human males were administered 1.0 μg pure AFB_1, and urine was collected over the next 10 days until no further AFM was detected. One subject excreted 5.6% of the initial AFB_1 as AFM_1 over a period of 5 days, whereas the second subject excreted 6.6% over a period of 7 days (Cheng et al., 1997). In a more realistic approach with regard to human exposure, Jubert et al. (2009) administered a single dose of 30 ng ^{14}C-AFB_1 to three volunteers and collected urine over 72 h. A total of 29–34% of the dose was excreted as AFB_1 equivalents during the study period; however, no information was provided regarding the amount of AFM_1 excreted. In a study of 617 children in high and low liver cancer incidence areas in China, the relationship between the dietary AFB_1 levels and AFM_1 excretion in urine was investigated (Tu, 1993). The conversion ratio from AFB_1 to AFM_1 excreted with urine was only 2.3% over 24 h.

Table 13
Regulated mycotoxins.

Substance	Abbreviation	IARC-classifi-cation	TDI-value (μg/kg b.w.)	Reference
Aflatoxins		Group 1[a]	–	IARC (1993)
Deoxynivalenol, 3-acetyldeoxynivalenol, 15-acetyl-deoxynivalenol	DON, 3-Ac-DON, 15-Ac-DON	Group 3	1.0 (gPMTDI)	FAO/WHO (2011)
Zearalenone	ZEN	Group 3	0.25	EFSA (2011a)
Fumonisins	FBs	group 2B	2.0 (gPMTDI)	FAO/WHO (2012)
Nivalenol	NIV	Group 3	1.2	EFSA (2013a)
Ochratoxin A	OTA	Group 2B	0.017	EFSA (2006)
Sterigmatocystin	STC	Group 2B	no TDI	EFSA (2013b)
T-2/HT-2 toxin		Group 3	0.1 (gTDI)	EFSA (2011b)
Citrinin	CIT	Group 3	0.2 (LNC)	EFSA (2012)
Patulin	PAT	Group 3	0.4 (PMTDI)	SCF (2000)
Enniatins	ENN		[b]	EFSA (2014)
Beauvericin	BEA		[b]	EFSA (2014)

gPMTDI: group provisional maximum tolerable daily intake; LNC: level of no concern for nephrotoxicity. IARC classification: group 1: carcinogenic to humans; group 2A: probably carcinogenic to humans; group 2B: possibly carcinogenic to humans; group 3: cannot be classified as to carcinogenicity to humans (IARC, 1993, 2002).

[a] AFM1 classified in IARC-group 2B.

[b] Insufficient data to establish a tolerable daily intake (TDI) or/and an acute reference dose (ARfD).

The overall intake could be backcalculated from the urinary excretion of AFM_1. In accordance to David (2000), we estimated the total daily intake (DI) using the equation

$$DI = \frac{C_{urine} * UV_{excr}}{F_{UE}}$$

where C_{urine} is the concentration of the metabolite in μg/l and UV_{excr} is the daily excreted urinary volume of 0.02 l/kg b.w. per day for adults (Kommission Human-Biomonitoring, 2007). F_{UE} is the molar fraction of the urine-excreted AFM_1 with respect to the parent compound. We used the mean F_{UE} value of 0.06 from Cheng et al. (1997). Regarding aflatoxins, no tolerable daily intake has been recommended so far. However, IPCS/WHO concluded that 0.01 cases of disease per 100,000 people corresponded to an intake of 1 ng/kg AFB_1 (see chapter 7). Although there is no EU legislation on the 'tolerable' risk level for carcinogens in society, ECHA stated in chapter R.8 that cancer risk levels of 1 per 10^{-6} could indicate tolerable risk levels for the general population (ECHA, 2012). This risk level is equal to an intake of 10 ng/kg b.w. AFB_1. Using the previously described equation and assumptions, a value of 30 ng/l of AFM_1 in the urine corresponds to the recent tolerable risk level discussed.

6.2. Ochratoxin A

Some groups used the general equation of Klaassen (1986) as a starting point to calculate the total daily intake (DI) via the concentration of OTA in plasma samples (Miraglia et al., 1996; Gilbert et al., 2001; Coronel et al., 2010). The DI can be quantified via

$$DI = \frac{Cl_p \times C}{A}$$

where Cl_p is plasma clearance in ml/kg b.w., C is the plasma concentration of OTA (μg/l), and A is the fraction of OTA that was absorbed from the gastrointestinal tract. Overall, the toxicokinetic data with regard to humans are limited and different assumptions of Cl_p and A are available in the scientific literature. First, renal clearance during the elimination phase in one human volunteer after ingestion of OTA was calculated to be approximately 0.048 ml/min or 0.99 ml/kg b.w. for a 70-kg person (Schlatter et al., 1996; Studer-Rohr et al., 2000). Secondly, Hagelberg et al. (1989) postulated a somewhat lower renal clearance of 0.033 ml/min in humans from animal data based on the rate of filtration in the glomeruli for inulin and the free fraction of OTA measured in human plasma. The relative bioavailability after oral exposure was 97% in mice, 44% in rats, and 57% in monkeys (Hagelberg et al., 1989). Schlatter et al. (1996) assumed a bioavailability of approximately 50% for humans. Taking into account the limitations of the previously mentioned data, the following equation can be expressed on the basis of human data:

$$DI = \frac{0.99 \times C}{0.5}, \text{ or}$$
$$DI = 1.98 \times C.$$

When using other assumptions based on the data given by Hagelberg et al. (1989), the remaining DI was approximately 30% lower.

We are able to compare the calculated total daily intake with toxicological values set by the EFSA in 2006 (see Table 13). At that time, a tolerable daily intake (TDI) of 0.017 μg/kg b.w. was established. If we used the equation described above and taking all limitations into account, the plasma concentration that corresponds with the TDI was 8.6 μg/l. Compared with the results from several surveys worldwide (see Table 10), this body burden was not reached in developed countries. For example, in a large study of 927 healthy volunteers in Germany, the 90th percentile and maximum value were 0.45 μg/l and 2.03 μg/l, respectively. Nevertheless, in some regions with endemic nephropathies or in some developing countries, some individuals show a daily intake above the TDI value.

6.3. Deoxynivalenol

Although free DON and its metabolites are rapidly excreted, these substances are generally suitable for human exposure studies and to assess the total daily intake from the levels observed in urine (Turner et al., 2010a; Warth et al., 2012a). The toxicokinetics of DON have been examined in several animal experiments, which concluded that approximately 28–75% of the orally administered dose was excreted as total DON (the sum of the free and glucuronidated forms) or total radioactivity in rats (e.g., Meky et al., 2003; Schwartz-Zimmermann et al., 2014; Wan et al., 2014; Guerre, 2015). In pigs, 43% of an applied single dose was recovered (Goyarts and Dänicke, 2005), and a compilation of 12 independent balance experiments suggested a strong linear relationship between DON exposure and the urinary excretion of metabolites (Dänicke and Brezina, 2013). In humans, Turner et al. (2010a) assessed DON (as well as its metabolites) in duplicate diet samples and in urine samples during periods of normal diet and wheat restriction interventions in 35 volunteers. They found a strong correlation between DON intake and the urinary biomarker. The overall average excretion of total DON via urine at the intervention phase was 72% of the ingested dose. In another experiment, one volunteer ingested (on the first and last two days) a mycotoxin-reduced diet but was given a naturally DON-contaminated diet during the four days between

(Warth et al., 2013b). Urine samples were collected for 24 h. The authors calculated an average daily excretion rate of 68% of the total DON. These results were further supported by a study analyzing DON in duplicate diets and in the excreted urine of one subject (Rodríguez-Carrasco et al., 2015). They found 72% of the total DON ingested in 24 hour urine samples.

The overall intake could be backcalculated from the urinary excretion of DON and its metabolites. In accordance with David (2000), we estimated the total daily intake (DI) using the following equation:

$$DI = \frac{C_{urine} * UV_{excr}}{F_{UE}}$$

where C_{urine} is the concentration of the metabolite in μg/l and UV_{excr} is the daily excreted urinary volume of 0.02 l/kg b.w. per day for adults (Kommission Human-Biomonitoring, 2007). F_{UE} is the molar fraction of the urine-excreted total DON metabolites with respect to their parent compound. We used the mean F_{UE} value of 0.70 of Turner et al. (2010a) and Warth et al. (2013b). A group provisional maximum tolerable daily intake (gPMTDI) of 1.0 μg/kg b.w. (see Table 13) was recommended for DON (FAO/WHO, 2011). Using the abovementioned equation and assumptions, a value of 35 μg/l urine of total DON corresponded with the recent gPMTDI. When these results were compared with those from biomonitoring studies (Table 12), a substantial percentage of the population was found to exceed the gPMTDI value.

6.4. Fumonisin B_1

Some toxicokinetic studies with experimental animals, such as rodents, monkeys, and swine, found that only 0.3–2% of the total FB_1 dose administered was excreted with urine (summarized in Van der Westhuizen et al., 2011). Regarding humans, only results from two studies using a more indirect approach have been published so far. In one study, 22 female participants from South Africa ate their traditional maize-based food, and urine samples were collected in parallel (Van der Westhuizen et al., 2011). On average, only 0.075% of the total FB_1 intake was observed in the urine samples. This very low value was supported by an additional study performed in the US (Riley et al., 2012). Here, 10 volunteers consumed 206 g/day of tortillas and biscuits prepared from masa flour and a product containing maize flour in different experiments over some days. The authors concluded that the average total urinary FB_1 was only 0.5% (from 0.12 to 0.9%) of the calculated intake. Accordingly, it appeared to be unreasonable to calculate the total daily intake from the excreted FB_1 concentrations.

6.5. Zearalenone

The metabolism of ZEN has been the subject of many studies in different species; nevertheless, toxicokinetic studies in animals and humans are limited. After a single oral dose of tritium-labeled ZEN, 45% of the radioactivity could be recovered in the urine of pigs (Biehl et al., 1993). Mirocha et al. (1981) studied the metabolism of ZEN in several experimental animals as well as humans. Here, an adult male was given a single oral dose of 100 mg, and the urine was collected over the following 24 h. ZEN and α-ZEL, primarily in their glucuronidated forms, were the main metabolites. Metzler et al. (2010) estimated from the previously mentioned study that after 24 h, approximately 10–20% of the dose was excreted as the parent compound in humans. In an additional experiment, Warth et al. (2013b) investigated the excretion profiles of one volunteer following a naturally contaminated diet containing 10 μg ZEN over a period of four days. They concluded that in this period on average 9.4% of the intake was excreted in the urine as total ZEN (the sum of the free and glucuronidated forms).

The overall intake could be backcalculated from the urinary excretion of ZEN and its metabolites. In accordance to David (2000), we estimated the total daily intake (DI) using the following equation:

$$DI = \frac{C_{urine} * UV_{excr}}{F_{UE}}$$

where C_{urine} is the concentration of the metabolite in μg/l and UV_{excr} is the daily excreted urinary volume of 0.02 l/kg b.w. per day for adults (Kommission Human-Biomonitoring, 2007). F_{UE} is the molar fraction of the urine-excreted total ZEN metabolites with respect to their parent compound. We used an F_{UE} value of 0.01 derived from the investigations of Mirocha et al. (1981) and Warth et al. (2013b). With regard to ZEN, a tolerable daily intake (TDI) of 0.25 μg/kg b.w. (see Table 13) was recommended (EFSA, 2011a). Using the previously mentioned equation and assumptions, a value of 0.13 μg/l urine of total ZEN corresponded with the recent TDI. Results from Italy, Belgium, Austria, and Germany were lower than 0.13 μg/l, whereas in other countries, especially developing countries, a significant percentage of the population exceeded this value.

7. Risk assessment/tolerable daily intake

EU legislation protects consumers by setting a framework of measures aimed at controlling foodstuffs and setting maximum levels for mycotoxins in food to ensure that they are not harmful to humans. Additionally, expert committees and institutions, such as the European Food Safety Authority, evaluate the toxicity of mycotoxins after considering relevant scientific information. At the end of a risk assessment process, toxicological values, such as the tolerable daily intake (TDI), are set. The TDI characterizes the total amount of a substance that can be ingested daily over a lifetime without appreciable health risks. Some TDI or risk values related to mycotoxins have been assessed by several institutions (summarized in Table 13). The total intake of some mycotoxins can be calculated and compared with these values. This approach is only applicable if sufficient toxicokinetic informations for a particular substance are available. Unfortunately, at present, these data are only available for a limited number of mycotoxins, such as OTA, DON, and ZEN.

Moreover, this approach has some limitations that must be discussed. For example, experimental and epidemiologic studies have consistently found sufficient evidence that AFB_1 is genotoxic in experimental animals and carcinogenic in humans and was consequently classified as carcinogenic to humans (group 1) (IARC, 2012). For this reason, no observed adverse effect level (NOAEL) could be established as a point of departure for risk assessment, and intake must be reduced wherever possible. Several studies in Asian and African countries reported consistent associations of aflatoxin intake and the development of hepatocellular carcinomas (HCC). Moreover, the potency of AFB_1 appears to be significantly enhanced in individuals with simultaneous hepatitis B infections (IARC, 2012). The majority of HCC cases (>80%) occur in developing countries; however, age-standardized HCC rates per 100,000 per year for females and males were 1.7 and 6.2, respectively, in Western Europe. Regarding cancer risks, IPCS/WHO estimated potencies and ranges for AFB_1 from epidemiological data and concluded that 0.01 cases/year per 100,000 individuals corresponds to an intake of 1 ng AFB_1/kg b.w. per day (range: 0.002–0.03) in hepatitis B-negative individuals (IPCS/WHO, 1998). In summary, Liu and Wu (2010) found a global burden of HCC that was attributable to aflatoxin exposure. They concluded that HBsAG-negative persons of the general population have a low risk (0.003–0.01 per 10^{-5}) in North America and (0–0.04 per 10^{-5}) Europe but a substantially higher risk (0.1–1.8 per 10^{-5}) in Africa.

OTA, FBs, and STC were classified by the IARC as group 2 B substances, which are potentially carcinogenic to humans. Comparing the total intake per day with tolerable daily intake values is important; however, potential cancer risks should also be discussed. OTA is a potent renal carcinogen in rodents. Notably, evidence from human studies remains inadequate, and the mechanism of tumor formation is under discussion (Clark and Snedeker, 2006, Haighton et al., 2012; Mally, 2012). The EFSA used a threshold-based approach in its risk assessment of OTA. They used the lowest observed adverse effect level (LOAEL) of 8 μg/kg b.w. due to the results noted for renal enzymes and renal function tests in pigs. They also applied an uncertainty factor of 450. Additionally, Kuiper-Goodman et al. (2010) estimated a negligible cancer risk intake (NCRI) using the tumorigenic dose at which 5% of the experimental animals are likely to have tumors (TD_{05}) as a point of departure. They concluded that daily exposure to 4 ng OTA/kg b.w. is associated with a risk of 1 per 10^{-5} (NCRI). Using Canadian data and a Monte Carlo simulation of mean-adjusted exposures, the NCRI was not reached by any age group, with the exception of 1–4-year-old children.

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