

Review Article

Diverse mycotoxin threats to safe food and feed cereals

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Toxigenic fungi, including *Aspergillus* and *Fusarium* species, contaminate our major cereal crops with an array of harmful mycotoxins, which threaten the health of humans and farmed animals. Despite our best efforts to prevent crop diseases, or postharvest spoilage, our cereals are consistently contaminated with aflatoxins and deoxynivalenol, and while established monitoring systems effectively prevent acute exposure, *Aspergillus* and *Fusarium* mycotoxins still threaten our food security. This is through the understudied impacts of: (i) our chronic exposure to these mycotoxins, (ii) the underestimated dietary intake of masked mycotoxins, and (iii) the synergistic threat of cocontaminations by multiple mycotoxins. Mycotoxins also have profound economic consequences for cereal and farmed-animal producers, plus their associated food and feed industries, which results in higher food prices for consumers. Climate change and altering agronomic practices are predicted to exacerbate the extent and intensity of mycotoxin contaminations of cereals. Collectively, this review of the diverse threats from *Aspergillus* and *Fusarium* mycotoxins highlights the need for renewed and concerted efforts to understand, and mitigate, the increased risks they pose to our food and feed cereals.

Aspergillus and *Fusarium* mycotoxins in cereals

Fungi are major crop pathogens and food spoilage moulds, which can contaminate our food and animal feed with an array of toxic secondary metabolites, referred to as mycotoxins [1]. Consumption of these mycotoxins is hazardous to humans and animals, with some mycotoxins being toxic at nanomolar concentrations [1,2]. Approximately 300–400 mycotoxins have been identified, where their consumption has been associated with cancers, neurological issues, and immune suppression [1]. Two prominent genera of mycotoxigenic fungi, *Aspergillus* and *Fusarium*, are responsible for the contamination of our major crops [3]. At present, mycotoxins produced by these genera are regarded as toxins of great importance and include aflatoxins, deoxynivalenol (DON), fumonisins, T-2 and HT-2 toxins, and zearalenone [4]. The opportunistic pathogens and food spoilage moulds, *Aspergillus flavus* and *Aspergillus parasiticus*, can both contaminate cereals (particularly maize) in the field and during storage, where they are known to produce aflatoxins, which are highly toxic and are classified as class I human carcinogens by the International Agency for Research on Cancer, in addition to causing digestive problems, plus reproductive and fertility issues [5–9]. Fusarium Head Blight is a key floral disease of cereals, primarily caused by *Fusarium graminearum*, a top ten fungal pathogen that produces mycotoxins such as DON and zearalenone, which reduces crop yields and grain quality, while posing a health risk [10–13]. The greatest risk of exposure to humans and farmed animals is through the consumption of mycotoxin-contaminated food or feed, which causes significant safety complications throughout our existing food and feed supply chains.

Of the global crop supply, an estimated 60–80% of crops are contaminated by mycotoxins, with 20% of crops exceeding the European Union (EU) legal food safety limits [14]. Cereals are a staple food, accounting for a global cropping area of 700 million hectares, which supplies approximately 40% of the energy

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and protein intake in a human diet [15]. Major cereal crops used for food and animal feed are particularly susceptible to mycotoxin contamination. In addition to threatening human health and food safety, mycotoxins cause significant economic costs, through their associated impacts on crop yields, animal productivity, and international trade [16].

Given the impacts on human and animal health, governments, such as the EU commission, set legal limits on permitted mycotoxin levels for food and depending on the specific mycotoxin, higher legal limits, or only recommendations, are set for animal feed [17]. Often, when mycotoxin levels are too great to be used for food, the cereal grains can be downgraded to animal feed; however, this comes at an economic cost to the cereal producer [18]. For example, 2 µg/kg aflatoxin and 750 µg/kg DON are permitted in unprocessed cereals for direct human consumptions, but 20 µg/kg aflatoxin and 8000 µg/kg DON are allowed in animal feed [19,20]. Both *Aspergillus* and *Fusarium* species can infect cereal crops in the field, inhibiting crop development, affecting grain production, and resulting in yield losses [8,13,21,22]. Aflatoxins contamination of crop seeds impacts crop growth and results in loss of market value [22]. *Fusarium* mycotoxins include DON, zearalenone, and fumonisins, can inhibit plant development by interfering with various metabolic processes. For example, DON has a negative impact on the growth of wheat seedlings [23].

To mitigate mycotoxins within our food and feed supply chains, governmental organisations such as the European Food Safety Agency (EFSA) and agribusinesses, such as (DSM BIOMIN, World Mycotoxin Survey) have established networks to monitor mycotoxin occurrence in food and animal feed [14,24]. Whilst these limits are effective at preventing acute human exposure to mycotoxins, the impact of *Aspergillus* and *Fusarium* mycotoxins still threatens farmed animal health, our economic sustainability, and the security of maintaining a safe supply of food and feed cereals. Understanding the different natures and diverse impacts of these hazardous fungal contaminants of cereals is vital in assessing how the associated mycotoxin threats may change in our future environments.

Multi-level economic and health impacts

Both *Aspergillus* and *Fusarium* species have broad host ranges that include important cereal crops. Specifically, maize followed by wheat, are the most frequently reported cereals with the highest concentration of aflatoxins and DON [25]. These mycotoxins can be metabolised, or partially detoxified, within their different plant or animal hosts, creating ‘masked mycotoxins’, which are more difficult to detect. The presence of these mycotoxins and their masked derivatives, within our cereals, have diverse economic impacts and health implications across the food and feed supply chains (Figure 1). Initially, the associated mycotoxin contaminations cause direct economic losses through the reduction of crop yields [2,4]. Harvested cereals that are not lost may then be lost indirectly, as fungal growth and mycotoxins reduce cereal quality and safety, resulting in them being downgraded to animal feed or outright rejected, where cereal farmers can face additional costs associated with waste disposal by landfilling or incineration [18]. Aflatoxin and DON contaminations can occur when cereals are either in the field or during storage. In the field, mycotoxin contamination is influenced by various factors, including the weather, crop variety, and insect damage. For example, elevated atmospheric CO₂ and crop growth at 20°C/18°C during the day/night cycle results in increased DON contamination [26]. However, aflatoxin is more commonly associated with postharvest contamination during grain storage, while DON is frequently reported as a preharvest in-field contamination issue.

Aflatoxins

Aflatoxin contaminations are predominately caused by *Aspergillus flavus*, which is ubiquitous in nature and agricultural areas [27]. Aflatoxin B₁ (AFB₁) is regarded as one of the most toxic agents to humans and farmed animals, causing weight loss, immunosuppression, growth impairment, and hepatocellular carcinoma formation [28] (Figure 2A). Specifically, malnutrition can occur when AFB₁ consumption results in hepatotoxicity through liver damage, interference with GH/IGF signalling and a reduction in hormone signalling, in addition to impacting nutrient absorption in the digestive system. However, the exact mechanism still needs to be fully understood in humans [29–31]. Within the livers of animals and humans, which have eaten AFB₁-contaminated food or feed, microsomal mixed-function oxidase enzymes (CYP1A2 and CYP3A4) convert AFB₁ into the hydroxylated-derivative AFM₁ and other metabolites, including AFB₁-8,9-epoxide (AFBO). AFBO and AFM₁ confer carcinogenic properties to AFB₁, where they conjugate on the N7 position of deoxyguanosine in DNA, forming an AFB₁-DNA adduct, 8,9-dihydro-8-(N7-guanyl)-9-hydroxy AFB₁ [32,33]. The adduct is unstable, promoting one of two things to occur on the guanine residue: (1) a stable open ring structure forms to make an AFB₁-formamidopyridine adduct (AFB₁-FAPy), or (2) depurination occurs, causing an increase of free AFB₁-N7-guanine [34]. Exposure to AFB₁, AFB₁-FAPy lesions and the AP site are all known to be associated with 249^{Arg} → 249^{Ser} mutations of the tumour-suppression protein p53 [35–37]. Therefore, AFB₁ consumption can result in increased DNA mutations, cell death, and liver cancer development [38,39].

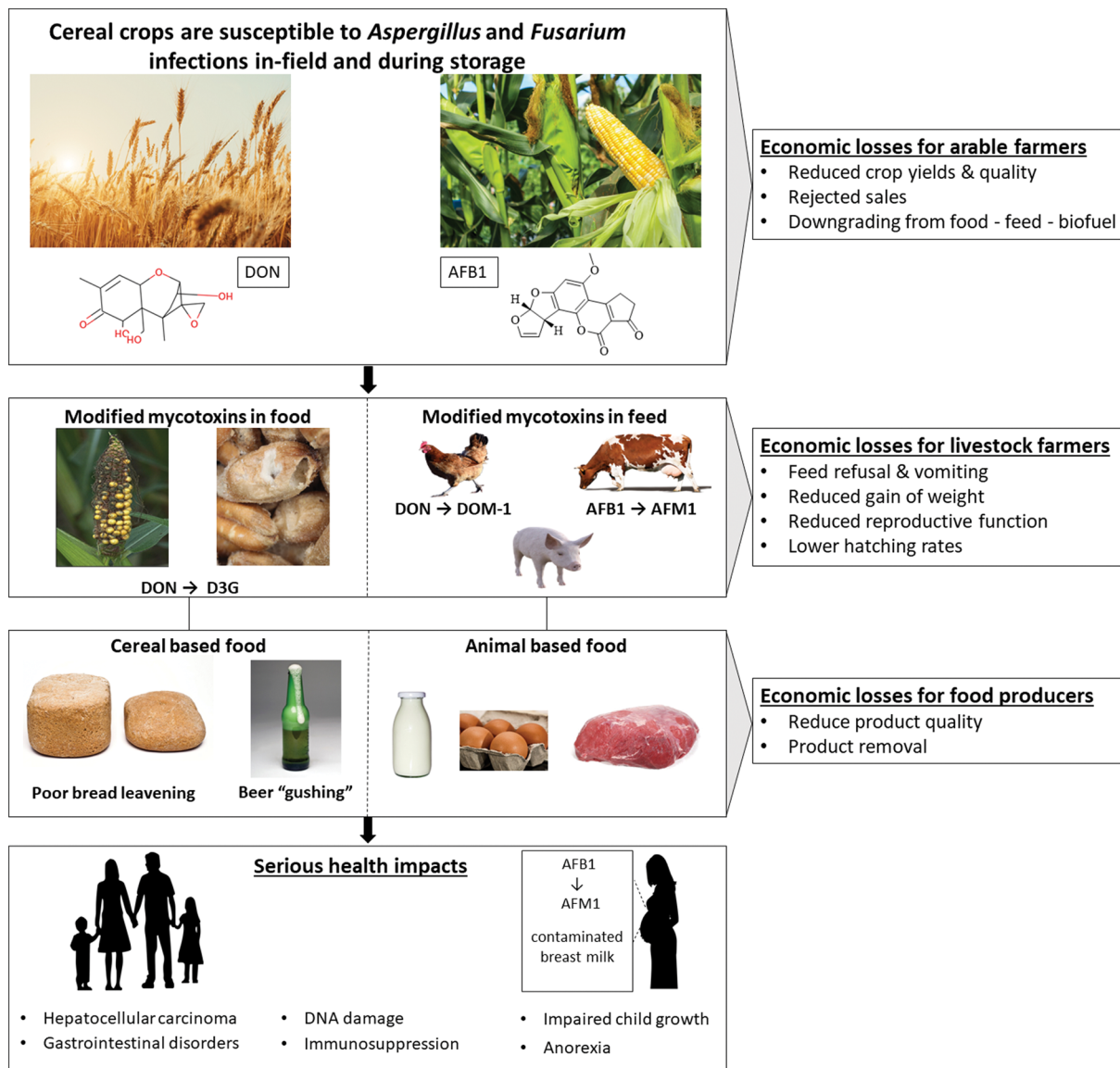


Figure 1. Economic and health impacts of mycotoxin in the food and feed supply chains

Highlights the impact of aflatoxin B1 (AFB1) and DON on cereal farmers and the associated issues for farmed animal producers. Ultimately cereal- or animal-based foods contaminated with these mycotoxins, or their masked derivatives, can seriously threaten human health.

Unsurprisingly, aflatoxin contamination of animal feed increases production costs, due to higher mortality rates, reproductive failures, reduced feed efficiency, and overall quality loss. Aflatoxins also impact the poultry industry, by delaying bird maturity, slowing body weight gain, decreasing egg production, and reducing quality, as eggs may contain AFB1 residues [40]. But egg quality is predominantly impacted via aflatoxins reducing the metabolic potential of the bird's liver, as proteins and lipids synthesized in the liver are incorporated into the egg yolks [41].

Aflatoxin-contaminated milk products are a particular concern, as cytotoxic AFM1 can be excreted in animal or human breast milk, where it is not denatured by heat or pasteurisation treatments [42]. Consequently, the consumption of AFM1-contaminated milk causes serious child health concerns, including stunted infant growth and increased susceptibility to infectious diseases [43]. AFM1 outbreaks dramatically affect the whole milk supply chain. One example comes from Serbia in 2013, where small farms were most affected, experiencing financial loss due to the lower milk prices they received from dairy companies, plus the cost of additional mycotoxin absorbers and milk testing. Collectively, these impacts lead to decreased demand and the reduced exportation of Serbian milk products [44].

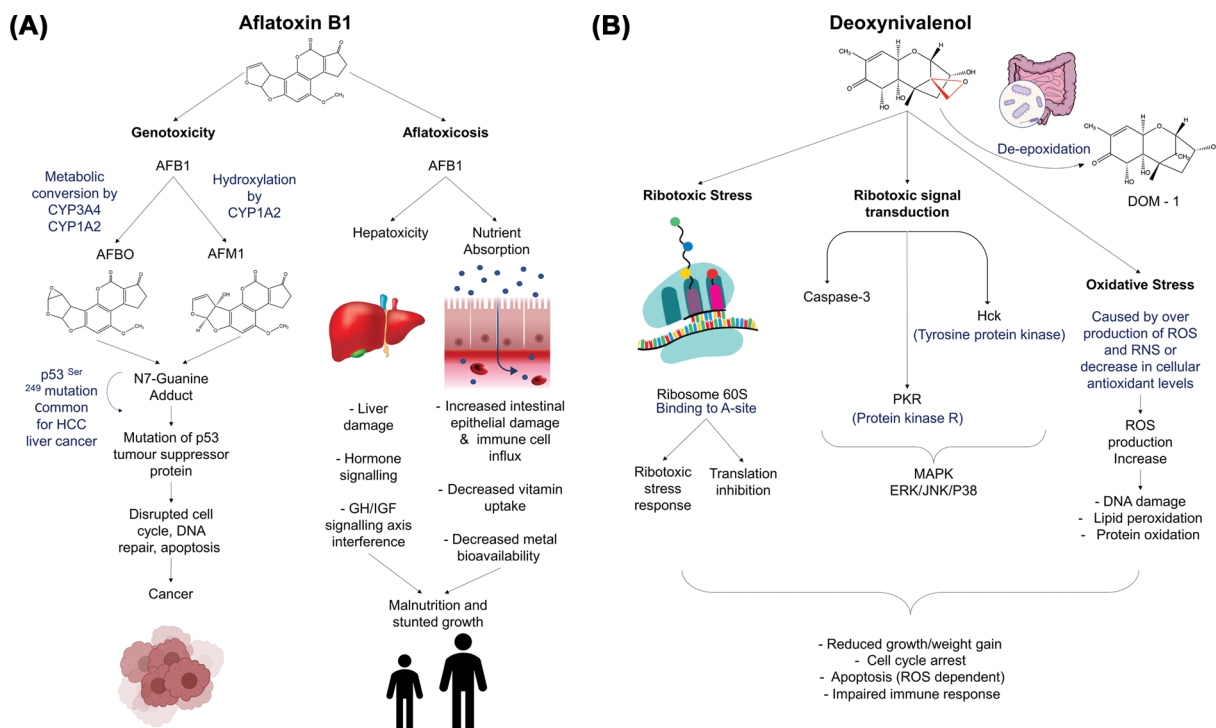


Figure 2. Mechanism of AFB1 induced cancer, malnutrition and stunted growth, and DON induced impairment of immune response, cell cycle arrest, and apoptosis

(A) AFB1 causes genotoxicity through the conversion and hydroxylation of AFB1 to AFBO and AFM1, resulting in mutation in the tumour-suppression protein p53. AFB1 additionally causes hepatotoxicity and interferes with nutrient absorption, resulting in cancer and growth reduction. (B) DON can be de-epoxidated into DOM-1 by gut bacteria that is less stable than DON and does not cause the ribotoxic stress response. DON can cause ribotoxic stress and interfere with translation inhibition. DON activates PKR and Hck resulting in increased MAPK. Oxidative stress is caused by increased ROS and RNS, and results in DNA damage, and protein oxidation whereby this leads to cell cycle arrest and apoptosis and impairment of immune response.

DON

Fusarium Head Blight and the associated mycotoxins, including DON, are prevalent throughout all cereal growing regions of the world. DON is a trichothecene mycotoxin and exerts its toxicity by binding to ribosomes – particularly the 60S subunit – impairing protein synthesis through the activation of cellular kinases associated with proliferation, differentiation, and apoptosis [45] (Figure 2B). DON can be modified through de-epoxidation by gut microbiota into DOM-1 [46]. However, DOM-1 which lacks the epoxy group, is not as stable when binding to ribosomes, and is therefore less toxic [47]. Upon consumption, DON targets innate immune system mononuclear phagocytes [48,49], causes the partial inhibition of protein translation, and activates ribotoxic stress. Translation inhibition can be caused by DON in several ways, such as interfering with the ribosomal peptidyl transferase function resulting in an impairment of initiation and elongation [50,51], through interfering with apoptotic associated pathways causing the degradation of 18S and 28S rRNA [52], and by inhibiting translation through the phosphorylation of eIF2α [53]. DON induces ribotoxic stress responses and triggers protein kinase R (PKR) and tyrosine-protein kinase, prior to mitogen-activated protein kinases signalling. However, DOM-1 does not activate the stress response due to the lack of a C12 hydrogen bond on the epoxy group and a hydrogen on uracil U2873 [47,54]. DON exposure also causes the accumulation of reactive oxygen species (ROS), through their overproduction and the decreased production of antioxidants. Increased ROS levels lead to cellular, nuclear and DNA damage, and protein oxidation [55]. Collective these outcomes to DON consumption impact on cell cycle arrest, apoptosis, and has effects on the immune system.

In wheat, DON causes the inhibition of immune responses enabling infection [56]. The consumption of cereal-based food and feed remains the primary route for DON exposure in humans and farmed animals [57]. In mammals, the consumption of DON-contaminated grain causes leukocyte apoptosis and immunosuppression, which in the case of farmed animals causes emesis, anorexia, poor growth, and development [51]. The pig industry is particularly susceptible, as pigs are unable to detoxify DON, resulting in poor growth that delays the time taken to reach

their marketable weight, which has serious economic consequences for producers [58]. Furthermore, the quality of edible animal tissues could pose further risks, as some studies have indicated that DON can be passed into animal products that could threaten human health if consumed [57].

Cereals crops can partially detoxify DON through its conjugation with a glucose moiety. This results in the generation of DON-3- β -D-glucoside (DON-3G). Specifically, this glycosylation changes the physicochemical properties of DON, enabling DON-3G to escape the extraction and detection methods commonly used to monitor DON contamination [59]. Although DON-3G is less toxic compared with DON, intestinal bacteria can hydrolyse DON-3G, releasing its toxic DON precursor during mammalian digestion. However, high concentrations of preintestinal bacteria present in ruminants and poultry can also convert DON into the nontoxic de-epoxide derivative DOM-1 [60]. Hence, dietary exposure to DON may be greater than estimated, where its masked forms may also present a serious threat to the safety of cereal-based food and feed, by contributing to elevating total dietary mycotoxin exposure [58].

Therefore, both cereal and farmed-animal producers, plus their associated food and feed industries, bear the costs associated with mycotoxins, which result in consumers experiencing higher food prices [61]. Importantly, the serious health threats from acute and chronic exposure to *Aspergillus* and *Fusarium* mycotoxins may not be disregarded. Understanding how these complex threats may change in our future agricultural environments and food supply chains is a priority.

Persistent mycotoxin threats to European farming and food safety

Analysis of the EFSA and BIOMIN records for mycotoxins detected over the last decade (2010–2020) shows the persistent risk of aflatoxins and DON in our main cereal crops (barley, maize, oat, rice, and wheat) in both our food and animal feed sectors, raising concerns associated with chronic exposure, masked toxins, and cocontaminations.

Aflatoxin and DON contamination of food cereals

DON was frequently detected in all food cereals, with 67.93% of maize and 48.16% of wheat contaminated (Figure 3). Of particular concern were the high average levels of DON found within maize (1448.1 $\mu\text{g}/\text{kg}$) and wheat (359.96 $\mu\text{g}/\text{kg}$), where 28.20% and 5.22% of samples had DON levels above the safety threshold (750 $\mu\text{g}/\text{kg}$) for human food, respectively, and 1.66% of maize was also above the guide for animal feed (8000 $\mu\text{g}/\text{kg}$) [19,20]. The exception was rice where only 3.89% contained DON and none was above the safe limit for human consumption.

Aflatoxin was also detected in all food cereals, but at a lower frequency than DON contamination, with 15.02% of maize and 11.79% of wheat being contaminated (Figure 3). At less than 5 $\mu\text{g}/\text{kg}$, the levels of aflatoxin contamination were also comparatively lower than DON contaminations. However, the safe food (2 $\mu\text{g}/\text{kg}$) and animal feed (20 $\mu\text{g}/\text{kg}$) thresholds for aflatoxin are much lower; therefore, aflatoxin contamination still results in 4.31% maize and 4.20% wheat being above the limit for food and 0.53% of maize also being above the limit for animal feed [19]. Contrasting DON contaminations where rice was not at risk, 10.79% of rice contained aflatoxins, with 0.95% of contaminations being above the safe food limit. Of all the cereals assessed, oats were least at risk of aflatoxin.

Therefore, our food cereals frequently contain tolerated levels of aflatoxin and DON, but this poses a health concern related to chronic mycotoxin exposure. Importantly, a significant proportion of food is consistently downgraded to animal feed, due to the detection of high contamination levels. For DON contamination across Europe in 2010–2019, Johns et al. (2022) estimated that 75 million tonnes of wheat (5% of food wheat) exceeded the 750 $\mu\text{g}/\text{kg}$ limit, where its downgrading to animal feed equated to a loss €3 billion [62]. Here, we showed that aflatoxins caused the downgrading of 4.2% food wheat between 2010 and 2020, potentially equating to an additional €2.5 billion lost. This clearly demonstrates the substantial economic impact of just two mycotoxins on cereal farming, an impact that was extenuated between 2021–2023, when food and feed wheat prices have risen from ~€200 to ~€300 per tonne. However, it also suggests the potential for our cereals to be cocontaminated with aflatoxins and DON at the same time, which could have largely uncharacterised human and animal health implications. Similarly, Johns et al. (2022) revealed that wheat is increasing cocontaminated with multiple different *Fusarium* mycotoxins, predominantly DON, zearalenone, and T-2 [62]. Therefore, greater understanding of the health implications of cocontaminations with multiple mycotoxins, and how these risks may change in future climatic scenarios is essential.

Aflatoxin and DON contamination of cereals for animal feed

High levels of aflatoxin and DON contamination in food cereals demonstrate that maize is particularly at risk (Figure 3). Comparisons of mycotoxin contaminations in EFSA and BIOMIN data show the different threats posed to our food and feed supply chains (Figure 4). Aflatoxin and DON contamination frequencies were only slightly increased

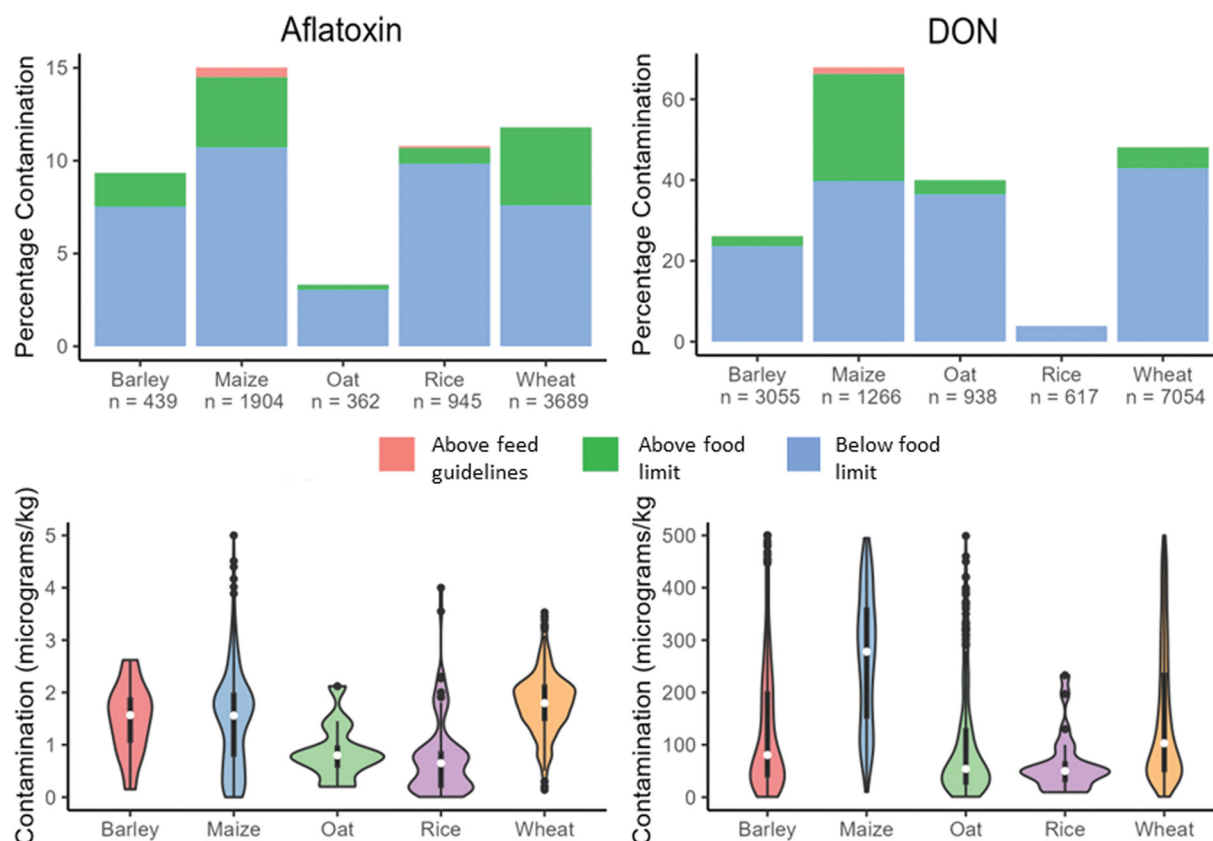


Figure 3. Contamination levels of food grains differ by grain for DON and aflatoxin

Contamination data are derived from EFSA records spanning from 2010 to 2020. The percent of contaminated samples below the regulatory limit for food (blue), above the food limit (green), and above the higher feed guideline/recommendation (red), with the total number of samples tested (*n*) for each cereal. Violin plots outlining the distribution of mycotoxin concentrations (μg mycotoxin per kg of grain) with superimposed boxplots displaying medians, interquartile ranges, and outliers. Due to a small number of extreme outliers for maize (as high as $790 \mu\text{g}/\text{kg}$ for aflatoxin and $32300 \mu\text{g}/\text{kg}$ for DON), y-axes were limited to the ranges shown.

in maize destined for animal feed. DON contamination levels were comparable between food and feed, around $\sim 750 \mu\text{g}/\text{kg}$ limit for human consumption [17]. However, at $\sim 15 \mu\text{g}/\text{kg}$, aflatoxin levels were significantly higher in maize destined for animal feed, which was significantly higher than the limit permitted for human consumption ($2 \mu\text{g}/\text{kg}$), dairy or young animals ($5 \mu\text{g}/\text{kg}$), and approaching the $20 \mu\text{g}/\text{kg}$ guide for other animals [4,58,63]. This again indicates the elevated risk of aflatoxin and DON mycotoxins in maize, while also demonstrating their diverse threats to our feed supply chains, where mycotoxins can undergo bioconversion into mixtures of masked toxins that are not easily detected using traditional testing systems.

How our changing agricultural environments influence the mycotoxin threat

Our agricultural environments are changing, and this is reflected in changing fungal crop disease pressures and mycotoxin contaminations. Driven by climate change and altered agronomic practices fungal pathogens are on the move, due to the geographic expansion of favourable conditions and hosts [64]. This can contribute to changing patterns in crop contamination, through the creation of increasingly favourable conditions for existing contaminations, or through the arrival of new mycotoxigenic fungal contaminants [62]. Next, we discuss how future environments may influence *Aspergillus* and *Fusarium* mycotoxin contaminations of cereals.

Optimal conditions for key fungal processes, namely mycelial growth, dissemination by sporulation, and mycotoxin biosynthesis, vary depending on the strain and nutritional environment assessed. However, the distribution of the outcome from published studies can reveal the optimal environments for *A. flavus* and *F. graminearum* (Figure

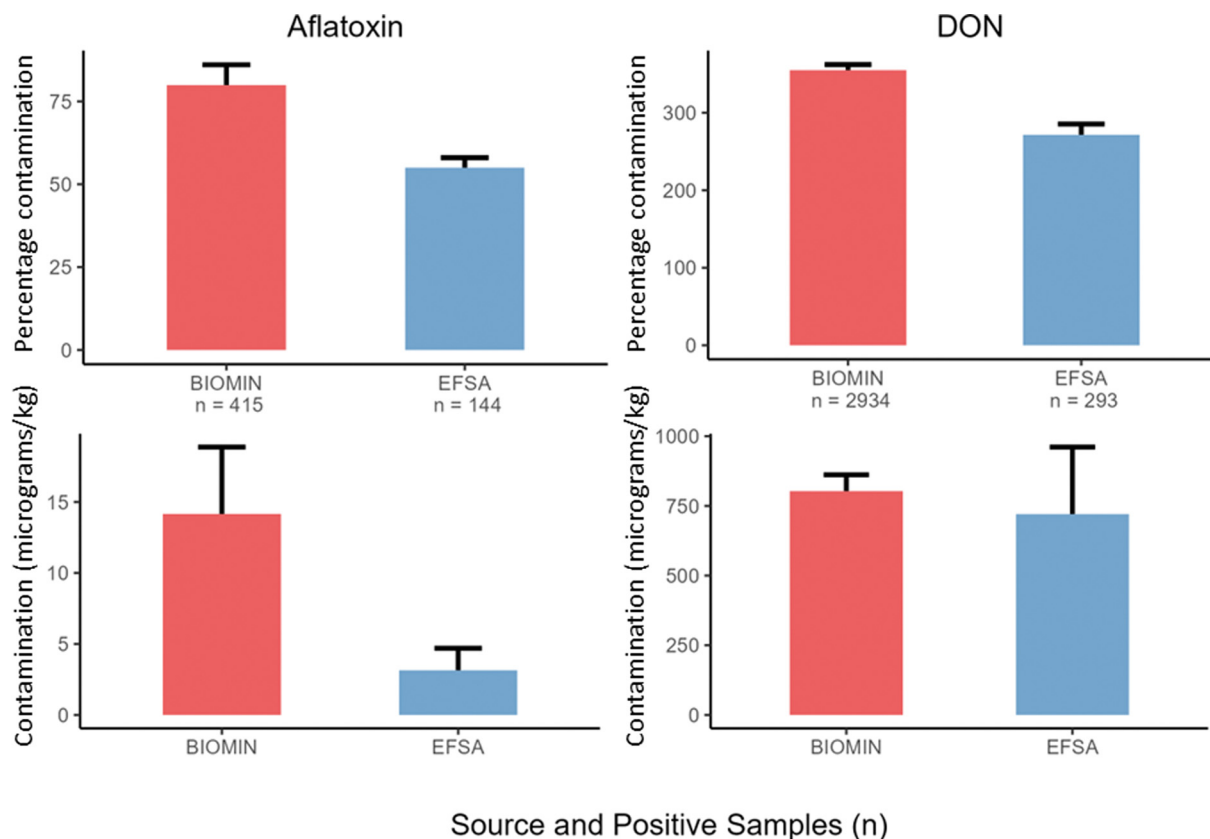


Figure 4. Mycotoxin contamination of animal feed maize exceeds contamination for food maize

Bar plots depicting the percentage and number (n) of samples containing aflatoxin and DON and the mean concentration (μg mycotoxin per kg of grain) of each mycotoxin. Contamination data for European feed maize from 2016 to 2022 were taken from BIOMIN GmbH annual mycotoxin reports and EFSA mycotoxin surveillance data were used for food cereals. Two outliers with excessively high contamination (790 $\mu\text{g}/\text{kg}$ aflatoxin, 23500 $\mu\text{g}/\text{kg}$ DON) were omitted from analysis. Error bars represent standard deviation around the mean.

5) and help us to understand how changing environments could lead to the increased risk of cereal infection and mycotoxin contamination (Figure 6).

Optimum T , a_w , CO_2 conditions for growth, dissemination, and mycotoxin production

A. flavus grows at temperatures from 10°C to 48°C [65]. However, the distribution of optima suggests between 28°C and 37°C best supports fungal growth (Figure 5). Unlike many other fungi, *A. flavus* is xerophilic and thus well adapted to grow at low water activity (a_w) with some strains growing at conditions as low as 0.73 a_w [66]. The abundant production and dissemination of airborne conidia and their subsequent germination is optimal at 25°C. Aflatoxin production also occurs at a wide range of temperatures, with an optimum between 28°C and 30°C [67]. Further to this, aflatoxins are predicted to become a major food safety issue in maize, especially in countries where a rise in temperature, together with increased CO_2 levels, and droughts are predicted to occur [68]. These traits allow *A. flavus* to thrive and outcompete other fungi in hot dry conditions, which can promote in-field cereal contaminations (Figure 6) or spoilage during storage.

The optimum growth temperature for *F. graminearum* is between 20°C and 25°C, with scant growth at low (5°C) and high (35°C) temperatures [69] (Figure 5), reflecting its geographic distribution across temperate-cereal-growing regions. The production of splash-dispersed asexual conidia favours slightly higher temperatures [10,70] with an optimum estimated at 28°C [71,72]. Similarly, perithecial maturation (20–25°C) and the discharge of wind-blown sexual ascospores (25–28°C) favour warm temperatures. This reflects the fine orchestration of sexual and asexual reproduction cycles with seasonal temperature changes [70] (Figure 6). DON production occurs over a narrow range

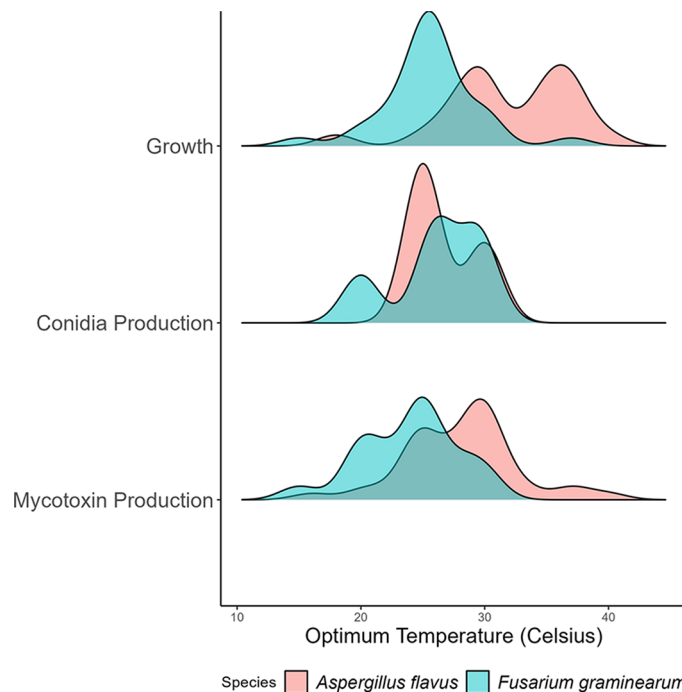


Figure 5. The optimum temperatures for key life processes for *A. flavus* and *F. graminearum*

Key life processes shown are the production of respective mycotoxins (aflatoxin and DON), mycelial growth in culture, and asexual conidia production. The density curves represent the distribution of published temperature optima. The height of a curve corresponds to the proportion of reports of optima at a given temperature.

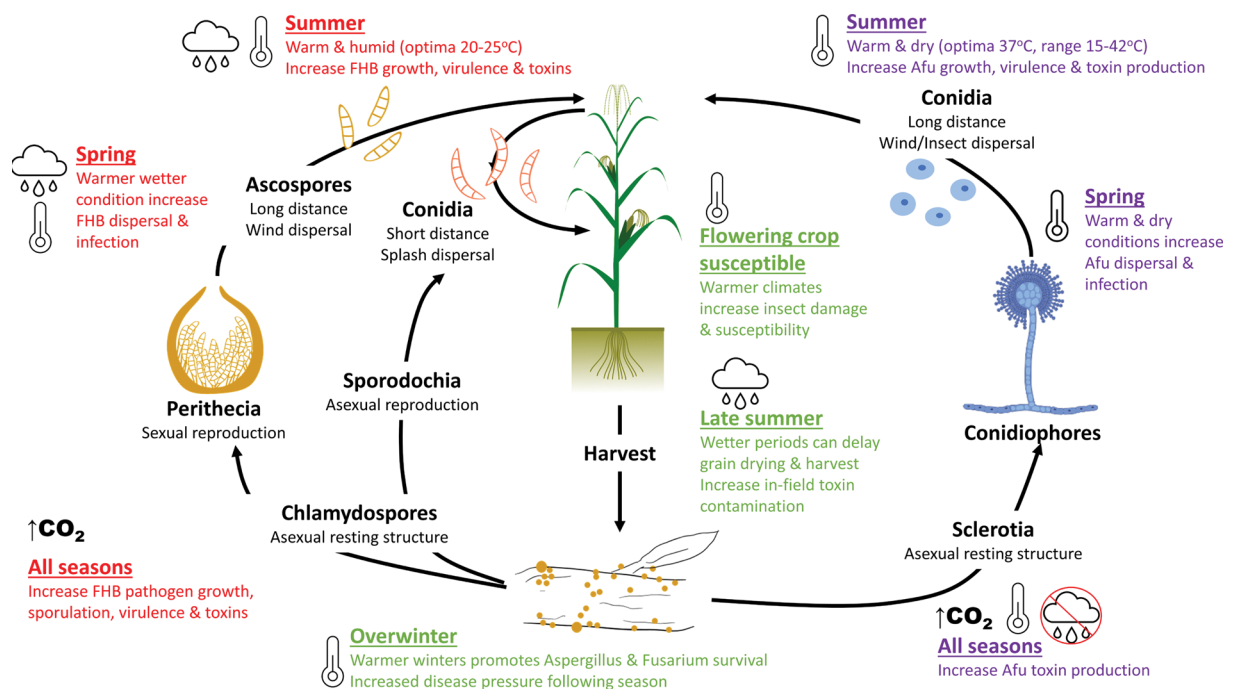


Figure 6. Climate change impacts on risk of cereal contamination by *A. flavus* and *F. graminearum*

Influence of climatic conditions on life process is shown in red (*F. graminearum*), purple (*A. flavus*), and green (both). Climate change is predicted to drive conditions in northern Europe towards warmer wetter environments favoured by *F. graminearum*. In contrast, southern Europe is expected to become hotter and dryer, as favoured by *A. flavus*. In both systems, elevated CO₂ is associated with increased mycotoxin contamination. Climate change projections may therefore allow for increased contamination of our cereals by mycotoxigenic fungi.

(15–30°C), with an optimum of 25–28°C [73,74]. As a hydrophilic fungus, *F. graminearum* thrives in wetter environments [75,76]. Mycelial growth only occurs above 0.9 a_w [66] with optima between 0.980 and 0.995 a_w [77]. Splashed-dispersed conidia intuitively require higher a_w than wind-dispersed ascospores and favour intense rainfall coinciding with crop susceptibility at anthesis [10,78]. Yet while ascospore dispersal can increase after a brief dry period, moisture also reliably predicts airborne ascospore prevalence, indicating that high moisture is indispensable for both ascospore and conidia dissemination [10]. DON production, as with temperature, occurs over a narrower a_w range (0.93–0.995 a_w) peaking at 0.995 a_w [66,73]. After fungal acclimation to the IPCC scenario predicted for 2100, elevated CO₂ was also found to promote cereal disease severity, impacting on seed number and weight [79]. Significant increases in DON contamination have also been observed in maize and wheat plants grown under increased CO₂ levels [26]. This may owe to greater host susceptibility, as elevated CO₂ has been shown to downregulate the defensive phenylpropanoid pathway in wheat [10]. These traits reflect why *F. graminearum* is a threat in warm moist environments, where it promotes in-field cereal contaminations (Figure 6).

How optimal conditions relate to Europe geographic mycotoxin threats

The two species presented illustrate how fungal pathogens occupy unique ecological niches. The optimum growth and mycotoxin production conditions depict clear differences, with *F. graminearum* favoured by warm wet and *A. flavus* by hot dry environments (Figure 6). Therefore, climatic changes may differentially influence the threat of *Aspergillus* and *Fusarium* mycotoxins across Europe.

In the last decade, *Fusarium* mycotoxins in cereals have increased, particularly in southern Europe [62], which may reflect the alignment of specific geographic environments with optimal conditions for contaminations to occur. However, European temperatures are expected to continue to increase by 3–5°C under climate change projections, with differing effects by region [80]. Southern Europe is predicted to experience increased droughts, while Northern Europe may see greater precipitation; with all trends projected to grow more severe [81]. The future warmer springs and increased precipitation in Northern Europe are likely to favour *Fusarium* outbreaks and mycotoxin contaminations [80], while climate change-induced warmer nights (+3°C) are conditions shown to increase *Fusarium* disease severity and DON production [82,83]. In contrast, as hot and dry episodes become more prevalent in southern Europe, this will favour increased *Aspergillus* infections and aflatoxin contamination. Climatic events, such as in northern Italy, have already been seen throughout the last decade in maize-growing regions prior to aflatoxin outbreaks [80]. Furthermore, outbreaks are predicted to become more of a threat with climate change, especially in the Mediterranean basin, which was highlighted to be warming 20% faster than the global average [84].

Climate change, therefore, threatens to exacerbate the intensity and extent of mycotoxin contamination in Europe, demanding concerted efforts in the surveillance and control of mycotoxigenic fungal pathogens, to mitigate the risks they pose to sustainable safe cereal production.

Summary

- Fungal pathogens, including toxigenic *Aspergillus* and *Fusarium* species, consistently contaminate our food and feed cereals, producing harmful mycotoxins, threatening human and animal health.
- Despite established monitoring systems and disease control approaches, mycotoxin contaminations consistently threaten to our food security, through the understudied impacts of: (i) chronic mycotoxin exposure, (ii) the underestimated dietary intake of masked mycotoxins, and (iii) cocontaminations with multiple mycotoxins.
- Mycotoxins have profound economic consequences for cereal and farmed-animal producers, plus their associated food and feed industries, which results higher food prices for consumers.
- Changing environments threaten to exacerbate the extent and intensity of *Aspergillus* and *Fusarium* mycotoxin contaminations of cereals, highlighting the need for concerted efforts to understand and mitigate increased risks.

Competing Interests

The authors declare that there are no competing interests associated with this manuscript.

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Author Contribution

R.L., J.B., A.B., and W.L. contributed to the analyses and wrote the manuscript. N.B. contributed to the conceptual design, funding, and preparation of the manuscript.

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Abbreviations

AFB1, aflatoxin B1; AFB1-FAPy, AFB1-formamidopyridine adduct; AFBO, AFB1-8,9-epoxide; DON, deoxynivalenol; DON-3G, DON-3-β-d-glucoside; EFSA, European Food Safety Agency; PKR, protein kinase R; ROS, reactive oxygen species.

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