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Review

Reducing the incidence of cereal head infection and mycotoxins in small grain cereal species

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ABSTRACT

Reducing the level of infection of cereal heads caused by *Fusarium* and associated mycotoxin accumulation in grains is of a high priority in order to secure the yield, agronomic performance, and food and feed safety from field to table. Strategies to tackle the problem have been proposed at many levels, including greater knowledge of the biology of toxigenic fungi and of plant–pathogen interactions, monitoring activities which extend from the field to the end-products, pre-breeding, breeding and transgenesis to develop new resistant plant varieties, crop protection based on synthetic or natural molecules, biocontrol of fungal populations, the development and use of models that take into account the climatic conditions, and the adoption of technological protocols for reducing or inactivating mycotoxins. This review article highlights that the problem is very complex but that the scientific community continues to produce important knowledge and potential solutions.

1. Introduction

1.1. Mycotoxins

Mycotoxins are secondary metabolites produced by several widespread moulds. These thermostable molecules, whose toxicity is normally chronic and only rarely acute, can be found as natural contaminants in many plants, particularly cereals but also fruits, hazelnuts, almonds, seeds, etc, and also in fodder, feed and food. At least 30 mycotoxins are truly toxic, with strong carcinogenic and genotoxic properties. The toxicity may vary considerably within a structural group of mycotoxins and the danger may not always be due to the toxin itself but also to its metabolites. A wide range of pathologies has been associated with mycotoxins, ranging from estrogenic, gastro-intestinal, urogenital, vascular, renal and neurological disorders to mutagenic, teratogenic and carcinogenic effects. Several mycotoxins can compromise the immune system, leading to greater susceptibility to diseases (Desjardins, 2006). Different mycotoxins can be present simultaneously because some moulds can produce more than one kind of mycotoxin or because of the simultaneous presence of different fungal species in raw materials, food or feed (http://www.efsa.europa.eu/en/scdocs/doc/22e.pdf), resulting in additive or antagonist effects (Speijers and Speijers, 2004). This is particularly true for forage that can be contaminated by mould flora which produce a large number of different mycotoxins. Moreover, contaminated crops, rejected for human consumption, can be in some markets be redirected to feed industry, resulting in the increased hazards in feeds and feed ingredients (Binder et al., 2007). In addition, an emerging issue is that mycotoxins can also be conjugated to other molecules, giving rise to soluble masked or bound mycotoxins, which can be metabolized by living plants, fungi and mammals or after food processing in free forms (see review by Berthiller et al., 2013). The conjugated forms are often less toxic than the free forms, therefore when assessing their toxicity the percentage of masked forms that can be hydrolyzed in the intestinal tract should be taken into account.

The presence of moulds on plants or foodstuff is not necessarily an indicator of the presence of mycotoxins, on the other hand the absence of visible moulds does not rule out their presence. Pre-harvest mycotoxins can be distinguished from post-harvest ones: the former are produced by fungi which invade their substrate and produce mycotoxins on the growing plants before harvesting. Aflatoxins and Fusarium-toxins are part of this group. The other group comprises the so-called post-harvest toxins, such as ochratoxin A, which is produced after harvesting and during crop storage and transportation. From an ecological and biological point of view it is not always clear why a fungus synthesizes mycotoxins, which are non-essential molecules for the viability of the cells. Reverberi

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et al. (2010) reviewed various hypotheses, for instance that mycotoxins may be one of the responses of the fungus to oxidative stress, to endogenous oxidants, and to the physiological changes induced by fungal differentiation. In addition, mycotoxins may have an ecological significance in that they give the toxigenic fungus an advantage over other organisms sharing the same trophic niche, for example through the inhibition of the quorum-sensing system of bacteria.

1.2. Toxigenic moulds

In recent decades there has been a global increase in events related to mycotoxigenic fungi (Goswami and Kistler, 2004), the main consequence being serious health risks both for humans and animals. *Aspergillus*, *Penicillium* and *Fusarium* moulds are most widely involved in mycotoxin production (Table 1). In the field, *Fusarium* spp. are highly successful pathogens, characterized by a variability in morphology and physiology which enable them to occupy diverse ecological niches in many geographical areas. Most plant species are susceptible to at least one disease caused by this fungal genus. *Fusarium* can colonize the xylem via the roots and the growing mycelium results in the obstruction of vessels and the prevention of water transport to the aerial parts (Leslie and Summerell, 2006; Di Pietro et al., 2003). In field, the plant–pathogen interactions that result in mycotoxin accumulation show a high level of complexity. Several different factors, ranging from the genetics of the plants and fungi to agricultural and technological practices, but also meteorological events, can affect the success or failure of the contamination. In addition to the health risks, significant yield reductions may occur in all major crops infected, with the yields of at least 25% of crops being affected annually.

1.3. Regulations

International and national legislation in many countries specifies maximum limits for several different types of mycotoxins in a variety of raw materials and foodstuffs (see http://www.mycotoxins.org/; http://www.efsa.europa.eu/en/topics/topic/mycotoxins.htm; van Hegmond et al., 2007). Additional more stringent limits are specified for several types of products for children and for pregnant and nursing women. However, surveys of estimated dietary exposure to the principal food mycotoxins show that cereals and cereal products are the main contributors to high exposure, due to their extensive consumption by almost all population groups, including adults, children, and vegetarians (Leblanc et al., 2005). Consequently, EU Regulations No. 856/2005 and No. 1881/2006 set out the maximum permitted levels of several *Fusarium* toxins in cereals and cereal-based products, becoming effective from 1 July 2006. These regulations are also updated whenever new or emerging mycotoxins are identified or there is new information on their potential toxicity, degradability etc. For example, the European Food Safety Authority (EFSA) recently published advice on human and animal health risks related to food and feed contamination with T-2 and HT-2 toxins (Schuhmacher-Wolz et al., 2010) and set the combined tolerable daily intake at 0.1 μg/kg bw/day. The European Commission subsequently published a recommendation for the continued monitoring of these mycotoxins and for investigations to identify factors that may cause excess contamination of grains by these toxins.

Table 1
The most significant mycotoxins in terms of diffusion.

Mycotoxin		Producer fungus
Aflatoxins	B1, B2, G1, G2	*Aspergillus flavus*, *Aspergillus parasiticus*
Ochratoxin A		*Aspergillus ochraceus*, *Penicillium verrucosum*, *Aspergillus niger*
Ergot alkaloids		*Claviceps purpurea*
Tricotecenes	Group A (T2–HT2, DAS)	*Fusarium sporotrichioides*, *Fusarium langsethiae*
	Group B (DON, NIV, 3-AcDON, 15-AcDON)	*Fusarium graminearum*, *Fusarium culmorum*, *Fusarium poae*, *Fusarium equiseti*
Zearalenone		*Fusarium graminearum*, *Fusarium culmorum*
Fumonisine	B1, B2, B3	*Fusarium verticillioides*, *Fusarium proliferatum*

1.4. Fusarium in small grain cereals

Fusarium species are necrotrophic pathogens that cause various diseases in cereals, such as root rot, foot rot, stem base rot, crown rot, Fusarium seedling blight, and Fusarium head (ear) blight (FHB) or scab. FHB is particularly destructive not only due to yield reduction but also because the occurrence of trichothecenes that bind to the 60S ribosomal subunit of eukaryotes, resulting in protein synthesis inhibition and apoptosis. An infected plant wheat has blighted heads and peduncle tissues which turn brown or tan and senesce prematurely. Visible symptoms of the fungus presence may include purple-black perithecia and/or pink sporodochia on heads, especially on glumes. The yield reduction is mainly due to kernel damage, resulting in wrinkled grains, with a characteristically bleached appearance and sometimes characterized by white or pink spots, or brown stains on the back of the caryopses (Sutton, 1982; Ruckenbauer et al., 2001). Table 1 reports the major fusariotoxins. The chemical structures of trichothecenes are characteristic of different *Fusarium* species, which facilitate chemotaxonomic identification.

In response to the important and complex problem of mycotoxin control, an impressive body of studies has been carried out to identify and understand the importance of factors involved in plant-pathogen interactions, including monitoring fungal epidemiology, management measures from the field to the final steps in the production chain, regulations or recommendations to limit mycotoxin content, the development of standardised analytical methods to determine mycotoxin content or fungal levels, decontamination by processing and general post-harvest strategies for mycotoxin removal. Some of these aspects will be considered here, focusing mainly on the control of FHB in small grain cereals.

2. Fusarium head blight (FHB)

FHB is mainly caused by *F. graminearum*, but other *Fusarium* spp., such as *Fusarium culmorum*, *Fusarium poae*, *Fusarium pseudograminearum*, and *Fusarium avenaceum* have been associated with FHB globally. The fungi can overwinter as saprotrophs in crop residues and their spores become dispersed during spring and summer. The fungal infection starts when the spores land on host plant tissues and germinate. The germ tubes develop into hyphae that can penetrate the plant tissue directly or can enter through natural openings or wounds. The final step is the sporulation. In wheat, the fungus invades floral tissues starting mainly from the anthers and palea and within a few days intercellular hyphae colonize the cortex cells of the rachilla and its vasculature. The hyphae invasion proceeds both inter- and intra-cellularly in the rachis node, the cortex and vasculature, and the fungus can therefore spread into the other spikelets. It has been hypothesised that colonization involves an initial phase, in which the fungus feeds off the extracellular exudates in the apoplast, and a necrotrophic phase, in which there is intracellular invasion that induces enhancement of jasmonic acid/