

Mycotoxins

The Institute of Food Science & Technology, through its Public Affairs and Technical & Legislative Committees, has authorised the following Information Statement prepared by its Professional Food Microbiology Group, dated February 2006.

SUMMARY

Mycotoxins occur widely in nature. There are several different types of these substances; all of them are produced by filamentous fungi. Organisms producing them can develop in foods at any stage in the food chain from the field to the plate. They can also enter the food chain by more indirect routes, for example, in milk from animals that have consumed contaminated material. Effective control requires a combination of good agricultural practice, carefully controlled storage and surveillance at every stage from field to plate. Developing economies are at particular risk from these contaminants as the (generally) moist, warm climates favour mould growth, while adequate control and good storage are difficult to achieve. This Information Statement presents a general overview of the problem, representing a basis from which more detailed Information Statements on specific Mycotoxins will be developed.

INTRODUCTION

Mycotoxins are natural products synthesised by certain filamentous fungi that cause a toxic response when introduced by a natural route (mostly by ingestion) in low concentrations to man and most other animals including birds and fish. Mycotoxicoses are possibly the most unfamiliar and least investigated of the diseases that affect man and domesticated animals (Smith *et al.*, 1994). At least twenty mycotoxins have, so far, been shown to occur naturally in foods and feeds at significant levels and frequency to be of a food safety concern.

Mycotoxins are produced by certain filamentous fungi growing naturally in many agricultural crops but especially in cereals including maize, wheat, barley, rye and most oilseeds in the field, after harvest and during storage and later when processed into food and animal feed concentrates (Smith & Henderson, 1991). Because of the relatively high intake of cereals and oilseeds in the diet of intensively farmed animals such as poultry, pigs and, to a lesser extent, cattle, there has been extensive documentation of the adverse effects on animal health and productivity when mycotoxin-contaminated feeds have been consumed (Berry, 1988; Smith & Moss, 1985; Smith *et al.*, 1994). Mycotoxins occur particularly in countries with climates of high temperature and humidity or where there is poor harvesting and storage conditions, which encourage mould growth and mycotoxin development. Human intake of mycotoxins occurs mainly from plant-based foods and from animal-derived foods such as milk, cheese and certain fermented meat products.

Human reactions to mycotoxins have been recorded for many centuries but only recognised as such more recently: the most significant is the disease known as ergotism. In this disease of rye, the fungus *Claviceps purpurea* grows in the developing seed and eventually replaces it with a

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hard fungal mass, not unlike the actual seed although slightly larger and darker in colour. When the rye seeds were harvested and ground into flour, the ergot was also ground, releasing a Pandora's box of deadly poisons *i.e.* ergotamine – a vasoconstrictor; ergonovine – a cause of spontaneous abortions; and ergine and lysergic acid hydroxyethylamide – causes of convulsions. In Europe in the Middle Ages, rye was the staple diet of the peasantry and records show that there were massive outbreaks of gangrenous and convulsive ergotism in the 9th and 16th centuries, and later, with huge levels of suffering. Thus, while for many centuries rye was the staple food, it was not only “the staff of life” but also “the sceptre of death”. It is widely accepted by most medical historians that, throughout the middle ages in Europe, the ergot mycotoxins exerted a major role in restricting population expansion and only the reduced dependency on rye cereal as the staple food in the 16th and 17th centuries, arising from the introduction of wheat and potatoes, allowed the steady upward movement in population growth. Other, more recent large-scale outbreaks of human mycotoxicoses, involving different mycotoxins, have occurred in India, China, and Russia, but in all of these outbreaks, including ergotism, there has been strong correlation with the consumption of *heavily* contaminated food sources.

Mycotoxins are, in general, low molecular weight, non-antigenic fungal secondary metabolites formed by way of several metabolic pathways, e.g. the polyketide route (aflatoxins), the terpene route (trichothecenes), the amino acid route (aflatoxin), and the tricarboxylic acid route (rubratoxin). Some mycotoxins, such as cyclopiazonic acid are formed from a combination of two or more of the principal pathways. While some mycotoxins are formed by only a limited number of fungal species, others can be produced by a range of species from several genera (Smith & Moss, 1985).

Mycotoxins normally enter the human and animal dietary system by indirect or direct contamination (Table 1). Direct contamination occurs when the food or feed becomes infected with a toxigenic fungus with subsequent toxin formation. Indirect contamination occurs when an ingredient of a process has previously become contaminated with toxin-producing fungi and, while the fungus may be killed or removed during processing, the mycotoxins will mostly remain in the final product. Most of the important mycotoxins are, in general, quite resistant to most forms of food and feed processing.

Mycotoxin contamination of food and feeds is a world-wide problem. It is now believed that as much as 25% of the world's food crops are affected annually by variable levels of mycotoxins, which can reflect substantial economic consequences for individual crops, livestock producers, grain handlers, processors, consumers and indeed national economies (Miller, 1998). In the US and Canada alone, losses to the feedstock and livestock industries due to mycotoxin occurrence has been estimated as US \$5 billion annually (Charnley *et al.*, 1995). Furthermore, in warm, humid developing countries, regular mycotoxin presence in the diet will also affect human populations, causing morbidity and premature deaths.

Direct economic losses resulting from mycotoxin-contaminated agricultural crops can be measured in reduced crop yields and lower quality, reduced animal performance and reproductive capabilities, and increased disease incidence. Such losses have been greatly underestimated in the past. Crop producers with mycotoxin-contaminated products will incur downgrading of crops, reduced markets, increased handling and processing, and increased costs for detoxification or dilution where these are legally permitted options. In the EU, it is illegal to blend food containing mycotoxins above the statutory maxima with material free of the toxins in order to reduce the concentration to a legal level. Also, it is illegal to treat foods containing

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aflatoxins by any chemical method in order to reduce the concentration of the toxin(s). Feed and food processors will also identify increased costs for further processing needs, especially for analyses and monitoring for presence of mycotoxins. Animal producers will identify increased production costs related to veterinary requirements, reduced outputs and possibly seeking new mycotoxin-free supplies. Methods for reducing or eliminating mycotoxins through the use of micro-organisms are now being developed. Reports include the application of fungi (particularly *Aspergillus* spp. related to toxigenic species) and lactic acid bacteria (LAB). A paper by Mokoena *et al.* (2005) describes using LAB to remove fumonisin B1 and zearalenone from maize meal. However, the legal position concerning these potential treatments is not yet clear, and it seems best to assume that they will be regarded in much the same light as existing strategies for reducing mycotoxin burdens below permitted thresholds, attractive though microbial upgrading of contaminated material may appear to be.

The populations of Western countries are undoubtedly less at risk than those of less developed countries since they are significantly protected by the high standards of the major food supplies and retailers, and the regulatory controls which deter the importation of seriously contaminated material. Sadly, as a consequence of such regulations, the most contaminated raw materials are still consumed by the local populations of the producer countries.

The main mycotoxins of practical interest include: the aflatoxins, ochratoxin A, the fumonisins, patulin, moniliformin, sterigmatocystin, the trichothecenes and zearalenone (see Table 2).

The many problems associated with this complex group of substances make it very difficult for any review to be complete (or even fully up-to-date). A recent text that tries to give such an overview is Magdan & Olsen (2004). The EU continues to work towards harmonised standards and the UK Food Standards Agency circulates regular progress reports on these discussions. Further details may be obtained from the FSA by following the link to “mycotoxins” on <http://www.food.gov.uk/aboutus/contactus/>

Among the many publications relating to these topics, Chapter 19 in the ICMSF *Microorganisms in Foods, 5, Characteristics of Microbial Pathogens* deals with “Toxigenic Fungi”, both broadly in the introduction to each section and, in considerable detail, listing all the properties relevant to food control (effects of pH, a_w , temperature, etc on growth and death) in both text and tables. It covers *Aspergillus parasiticus*, *A. flavus*, *A. ochraceus* and related species, *A. versicolor*, *Fusarium equiseti*, *F. graminearum*, *F. moniliforme* (this organism is now classified as *F. verticilloides*; while this now constitutes the correct systematic name, most industrial workers concerned with this group of infestations continues to use the older name, so for simplicity it is retained here, but with a strong recommendation to change to using the current name), *F. sporotrichoides*, *Penicillium citreonigrum*, *P. citrinum*, *P. crustosum*, *P. islandicum* and *P. verrucosum*.

For the food and related industries, satisfactory sampling of raw and processed materials is a particular problem. This is addressed in another ICMSF publication, No. 7 of the same series, *Microbiological Testing in Food Safety Management* (2002), in which Chapter 7 deals with “Aflatoxins in peanuts”.

In a field that develops as rapidly and is as diverse as that of mycotoxins, it is axiomatic that anything is out of date as soon as it is published. Thus, this Information Statement can at best be a “benchmark”, trying to present the position at a particular point in time. There are sources,

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particularly on the web, that may assist workers in addressing this dilemma. One site that may be found very useful is the European Mycotoxin Awareness Network, available at <http://www.mycotoxins.org> which provides information and news on many areas, such as HACCP / prevention, sampling (again, see previous paragraph), legislation (of particular importance in the UK context because of the extent to which we are now subject to EU legislation) and surveillance. The inclusion of fact sheets about the individual mycotoxins further increases its value.

HEALTH IMPLICATIONS OF MYCOTOXINS

There is now extensive documentation confirming the wide range of adverse effects on animal health and productivity when mycotoxins, especially the aflatoxins, are present in the diet (Smith *et al.*, 1994; Smith and Henderson, 1991). Animals can demonstrate variable susceptibilities to mycotoxins, depending on genetic factors (species, breed and strain), physiological factors (age, nutrition, etc.) and environmental factors (climatic conditions, husbandry and management). Multi-mycotoxin exposure in compound feeds presents special difficulties. In most developed countries, the natural contamination levels of mycotoxins in animal feeds do not normally occur at levels that can cause acute or overt mycotoxicoses, e.g. hepatitis, haemorrhage, nephritis and necrosis of oral and enteric epithelia, and even death. Rather, the observed levels induce symptoms of chronic primary mycotoxicoses and immune suppression. It is often difficult to observe or diagnose such manifestations of disease but certainly they represent the most common forms of mycotoxicosis in farmed animals, e.g. reduced productivity in the form of slower growth rates, reduced reproductive efficiency, inferior market quality, reduced feed conversion efficiency, reduced milk yields or reduced egg production. It is not yet possible to demonstrate such chronic primary mycotoxicoses in humans. However, there is continuing concern that populations subjected to regular exposure to low levels of mycotoxins could have similar adverse symptoms.

Resulting from their diversity of chemical structures and differing physical and biochemical properties, mycotoxins exhibit a wide array of biological effects on mammalian systems, including man, and individual mycotoxins can be genotoxic, mutagenic, carcinogenic, embryotoxic, teratogenic or oestrogenic (for further details, see Smith & Henderson, 1991).

The effects of mycotoxins on human health are complex and, mostly, little understood. Human mycotoxicoses have been mainly associated with the ingestion of contaminated foods, although the respiratory route, via the inhalation of spores, mycelial fragments and other airborne particles (particularly agricultural materials), cannot be discounted. For example, high levels of aflatoxins (c. 200,000 µg/kg) have been determined in both conidia and sclerotia of *A. parasiticus* and *A. flavus*, while spores from other toxigenic fungi have been shown to carry the toxin(s) relevant to the fungus (Lewis *et al.*, 1993). Many toxigenic species have spores capable of reaching the alveoli of the lungs and, because of the solubility and low molecular weight of most mycotoxins, such toxins are likely to be absorbed via the respiratory epithelium and translocated to other sites (Flannigan *et al.*, 1991). However, it is the gastrointestinal route of toxin absorption associated with contaminated foods that continues to attract the most attention (Smith and Henderson, 1991).

Improved agricultural practices and better storage and transportation facilities have reduced toxigenic mould growth in the raw materials used in the human food chain. However, there are continued concerns on the possible adverse effects resulting from long-term exposure to low

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levels of certain mycotoxins (especially aflatoxins) in the food chain (Smith *et al.*, 1994). There continues to be insufficient awareness of the effects of varying levels of ingestion of a single mycotoxin or a mixture of mycotoxins, the susceptibility of individuals and the effects of other types of toxins in the environment on synergy or even on antagonism. The effects are well demonstrated in animal systems through careful experimentation, yet the food industry continues to underestimate their undoubted presence (with the exception of the aflatoxins) within the human food chain. In large part, this can probably be attributed to the complex sampling and analytical problems associated with these substances, and it does not reflect on the food industry's concern for these contaminants. Although a Food Standards Agency survey (2002) identified elevated levels of certain mycotoxins in some retail samples of nuts and nut products in the UK, a further survey in 2004 showed a marked improvement, with the level of aflatoxins in the majority of samples (70%) being so low that they were not quantifiable, while a further 25% of the samples contained aflatoxins but below the regulatory limits. None of the nut product samples exceeded the regulatory limits. The FSA stated at the time "the consumption of a very small amount of a mycotoxin on a single occasion, such as consumption of a small amount of contaminated nuts, is unlikely to cause ill effects". However, little is known concerning the consequences of mycotoxin intake on what would be considered as the most vulnerable individuals such as children, pregnant women, the aged, the immuno-compromised and individuals who are receiving a nutritionally inadequate diet.

Most of the available understanding of modes of action, pathological conditions and degrees of toxicity of mycotoxins has been derived from experimental studies on animals other than man. Genotoxicity resulting from carcinogenicity, mutagenicity and/or teratogenicity has long been determined with the aflatoxins and clearly demonstrated in a wide range of animal species (IARC, 1993). It is now accepted that aflatoxins are undoubted human carcinogens. The potential carcinogenicity of sterigmatocystin, ochratoxin A, T-2 toxin, patulin, griseofulvin and zearalenone is now accepted for animals and highly suspected in humans.

It is extremely complicated to establish a causal relationship between mycotoxin exposure and human disease because of the uncertainties associated with human epidemiological studies. The linkage between mycotoxin exposure and human disease is now being studied by way of biomarkers for aflatoxin and other mycotoxins. This relies on the knowledge of the metabolism and macromolecular adduct formation in urine, blood and milk to estimate the exposure of individuals to such mycotoxins and then predicting the risk of developing cancer and other diseases (Coker, 1997; Wild *et al.*, 1988). Where there is a high expectancy of aflatoxin in the human diet, such as warm, humid developing countries, it is strongly implicated in the aetiology of kwashiorkor (the condition normally regarded as a consequence of protein insufficiency in the diet, especially of growing children), and it is present in human breast milk, the blood of pregnant women and babies' cord blood at birth (Smith *et al.*, 1995, for references).

Strong scientific evidence confirms that several mycotoxins have been shown to damage components of the human immune system involving cellular immune phenomena and non-specific humoral factors associated with immunity (Pestka and Bondy, 1994). Indeed, it is now strongly accepted by many mycotoxicologists that the most important single toxic role of many mycotoxins on human health may well be the insidious effects on the human immune system. Infectious diseases such as respiratory infections, diarrhoeal diseases, tuberculosis, measles, HIV and others, are amongst the main killers of children in developing countries (Miller, 1998). A World Bank report has strongly incriminated mycotoxins (especially aflatoxins) as a significant modulating factor in these childhood diseases and deaths (World Bank, 1993).

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Continued understanding of the interactions between mycotoxins and cellular components will be required to fully understand the mode of action of mycotoxins. It is also important to note that some mycotoxins can act directly on cellular systems while others must be converted into active forms. In the final summation, the toxic response by a mycotoxin will be critically influenced by the rate of absorption, distribution, biotransformation and excretion (Smith *et al.*, 1994).

Risk assessment of mycotoxins is now an advanced science and such studies serve as the basis for their risk management (WHO, 1995; Kuipers-Goodman, 1998). Epidemiological studies and animal toxicity studies continue to contribute the greatest predictive information and this is being augmented with the use of *in vitro* assays with human cell lines (Lewis *et al.*, 1998).

In most developed countries, where low exposure levels are the general rule, mycotoxins will represent a chronic hazard. Because of the confirmed or suspected carcinogenicity of several mycotoxins, they must be considered to be among the highest of any diet related risk to humans when compared with other microbial toxins, phycotoxins, food additives and pesticide residues. This insidious aspect of mycotoxins needs to be more widely appreciated by food microbiologists and the food industry.

REGULATORY LIMITS FOR MYCOTOXINS

Throughout the world, there are many advisory bodies concerned with food safety, including the Joint FAO / WHO Expert Committee for Food Additives (JECFA) and the European Food Safety Authority (EFSA), which regularly assess the risk from mycotoxins and advise on controls to reduce consumer exposure (e.g. MAFF, 1999; FAO, 2001; Codex Alimentarius, 2002). The European Mycotoxin Awareness Network is an establishment funded by the EU and currently involves 13 countries; its UK contacts are Cranfield University Biotechnology Centre and Leatherhead Food International. Its website is indexed in the Bibliography. In the UK, the Food Standards Agency is now responsible for ensuring mycotoxin safety (Food Standards Agency, 2002). UK legislation on mycotoxins is harmonised with the EU but, additionally, guidelines exist for Patulin (50µg/kg).

Regulation (EC) No. 466/2001 sets the basis for EU controls on residues and contaminants, and has already been amended several times. Maximum levels for aflatoxins are currently set at 2µg/kg Aflatoxin B₁ and 4µg/kg total aflatoxins in a range of products for direct human consumption.

Under Regulation (EC) No. 472/2002, maximum limits for Ochratoxin A (OTA) are set at 5µg/kg in raw cereal grains, 3µg/kg in all products derived from cereals (including processed cereal products and cereal grains intended for direct human consumption) and 10µg/kg in dried vine fruits (currants, raisins and sultanas).

Regulation (EC) No. 123/2005 extends the scope of OTA controls and establishes maximum levels of OTA in roasted coffee (5µg/kg), soluble coffee (10µg/kg), wine (2µg/kg) and grape juice and musts (2µg/kg). It also includes maximum levels for baby foods, cereal-based weaning foods and certain infant foods for special medical purposes (0.5µg/kg).

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In addition, Regulation (EC) No. 856/2005 will establish maximum levels for the *Fusarium*-toxins Deoxynivalenol, Zearalenone and fumonisins from June 2006, and foresees maximum levels for T-2 and HT-2 toxin in cereals and cereal products from 2008 onwards.

A proposed EU Recommendation on the prevention and reduction of *Fusarium* toxins in cereals and cereal products is in the final stages of preparation.

ANALYTICAL METHODS OF SURVEILLANCE

Efficient sampling, sample preparation and methods of analysis are the continuing aims for identifying the extent of mycotoxin presence in food, feed ingredients, human and animal tissues, blood, urine and milk to supply the fundamentals for control procedures (Smith *et al.*, 1994; Steyn *et al.*, 1991). The main analytical procedures for isolating the major mycotoxins (especially the aflatoxins) from complex biological matrices and the ensuing separation and purification follow well-established flow patterns, viz. sampling, extraction, clean-up, separation, detection, quantification and finally confirmation. Although there are now well-established protocols for each stage of these analytical procedures which have been adopted and incorporated into legislation, modifications are regularly being devised to achieve more accurate and reproducible identification of individual and multiple mycotoxins in natural products and mammalian body fluids (CEN, 1999; EU Commission, 2002; Mélotte, L, 2004).

A cardinal feature of any analytical process for mycotoxins is the design and efficacy of the original sampling plan, since this is almost always impeded by the highly positively-skewed distribution of the mycotoxins in the raw agricultural products that are being analysed. All product sampling steps should be carried out as accurately as possible, so that the final samples that will be chemically analysed are truly representative of the batch under examination. Inappropriate or biased samples will obviously invalidate the resultant analysed data. The complexities associated with the design of acceptable sampling protocols for most mycotoxins have been comprehensively reviewed by Coker *et al.* (1995), who have examined these differing but complementary approaches, viz:

- The use of theoretical probability models
- The use of statistical models to express the variance of the sampling test results
- Sampling plans for problematic commodities.

Mycotoxin purification from commodities has been greatly advanced in recent years by the use of solid phase extraction columns, which are quick, solvent-efficient and economical. The analyte can be eluted into a small solvent volume to be injected into a liquid chromatograph, with possible automation for large-scale sample handling. For the aflatoxins, reverse phase liquid chromatography is increasingly being used and pre-column derivatisation with trifluoroacetic acid has been used for several commodities after clean-up using multi-functional filtration column. Such multi-functional clean-up columns provide an extremely rapid removal of interferences from food extracts by lysophilic and charged sites present in the proprietary packing materials. The aflatoxins will pass through the column and can be quantified by HPLC with pre-column or post-column derivatisation (Wilson & Romer, 1991).

The availability of well-characterised monoclonal antibodies has had a major influence in establishing immunochemical methods for mycotoxin analyses, especially with the aflatoxins. The use of such specific antibodies in competitive enzyme immunoassays, either as microtitre

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plate assays or membrane-based quick tests, as well as for the production of immuno-affinity columns is now widely practiced especially for total aflatoxins, aflatoxin B₁ and aflatoxin M₁. Such immunological methods are increasingly been used at different stages of an integrated analytical system for mycotoxin determination in feed and food systems. Aflatoxin-specific antibodies in clean-up columns are now widely used as a substitute for conventional physico-chemical methods of clean-up, allowing them a direct qualification by TLC and HPLC (Smith *et al.*, 1994).

The development and improvement of analytical methodology and sampling plans for several mycotoxins has been greatly enhanced by the increased availability of matrix matched, certified reference materials (CRMs). The type of matrix CRMs and concentration of the mycotoxin are based on the natural occurrence patterns of the mycotoxin in specified foods and feeds. The availability of suitable CRMs, while being a prerequisite for the implementation of regulations and standards, will also be invaluable in many ways for the validation of new methods, solving trade disputes and for harmonising proficiency schemes (Boenke, 1995).

While extremely low permissible levels of mycotoxins are set by legislation and advisory guidelines, there is considerable evidence of the difficulties encountered in, for example, aflatoxin sampling and analysis. Such analyses require considerable skill and attention to achieve consistent results, and while consistency can be usually achieved within an individual laboratory, there still exists considerable inter-laboratory variability when analysing at threshold limits. Most current limits are set close to the limits of performance of available analytical methods (Horwitz *et al.*, 1993).

PREVENTION AND CONTROL OF MYCOTOXINS

Mycotoxins primarily enter the human and animal food chains through agricultural products, mainly cereals, nuts and oilseeds or from products later derived from these sources (Smith and Henderson, 1991). While mycotoxin contamination of seeds is mostly caused by inadequate storage conditions of harvested crops, pre-harvest contamination of the seeds can also occur especially with *Fusarium* spp. producing zearalenone, the trichothecenes and fumonisins, while other fungal contamination can produce ergot alkaloids, tremorgen mycotoxins and aflatoxins.

Preventative treatments to reduce pre-harvest fungal penetration of seeds and consequent mycotoxin formation have included breeding fungal resistant crop plants; good agronomic practices; harvesting the crop at the optimum stage of development; rapid reduction of moisture level by correct drying; and application of pesticides (Lisker & Lillehoj, 1991).

Post-harvest contamination by mycotoxigenic fungi usually occurs during storage and transportation and is normally caused by improper drying or re-wetting of the crop from rain or condensation. Basically, effective post-harvest management technologies involve correct drying followed by efficient storage and monitoring. By far the most critical environmental factors determining whether a product will support mould growth are temperature, moisture content and time, and each of these parameters can be controlled for the prevention of mould growth (Smith & Moss, 1985).

When physical methods of preservation are not successful, increased uses of chemical preservatives become essential. When used at correct internationally-agreed levels, these compounds can successfully restrict the growth of mycotoxin-producing fungi. Various organic

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acids such as sorbic, benzoic, propionic, acetic and formic, have been widely used as preservatives and are normally used as the corresponding sodium, potassium or calcium salts.

However, when mycotoxins have been identified in an agricultural crop, e.g. peanuts, corn, cottonseed, what can be done? There are only two basic approaches, viz:

- The mycotoxin contaminated product can be destroyed or the individual contaminated seeds removed.
- Subject to local legislation, the mycotoxin can be degraded into less-toxic or non-toxic products.

Various techniques have been devised, based on colour and visual appearance of decay or damage to separate out contaminated seeds (usually peanuts and other nuts). This may be manual or by more advanced electronic instrumental selection (West & Bullerman, 1991).

Where they are legally permitted, detoxification or decontamination processes will involve degradation, destruction and/or inactivation of the mycotoxin. Detoxification, while reducing the concentration of the mycotoxins to safe levels, should not produce toxic degradative products or reduce the nutritional or palatability properties of the commodities. Whilst many methods have been attempted, especially for the aflatoxins, only a very few offer some hope for practical application. The main potential candidate is ammonia, either in the gaseous form or as an ammonium hydroxide solution, used at various temperatures, pressures, moisture content and reaction times to degrade aflatoxins in various animal feedstuffs. Aflatoxin levels can be reduced by up to 99% (Coker, 1997).

In recent years, there have been extensive studies using phyllosilicate clays (hydrated sodium calcium aluminosilicate) to chemi-sorb aflatoxin in aqueous suspensions, including milk, reducing the uptake of aflatoxin by blood and its distribution to body organs such as the liver, reduce the transmission of aflatoxin M₁ to milk in lactating dairy goats and cattle, and to decrease the toxic effects of aflatoxin to many animal species (Phillips *et al.*, 1995; Galvano *et al.*, 2001).

The awareness of the toxic role of mycotoxins in the human diet would be greatly improved by the wider teaching of mycotoxicology in courses on food science, nutrition and microbiology.

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Table 1: Probable routes for mycotoxin contamination of foods and feeds

Mould damaged foodstuffs	
Agricultural products, e.g.	
Cereals	} Major source
Oilseeds	
Fruits	} Minor source
Vegetables	
Consumer foods (secondary infections)	
Compounded animal feeds (secondary infections)	
Residues in animal tissues and animal products	
Milk (animal and human)	
Dairy produce	
Meats (liver, kidney)	
Mould-ripened foods	
Cheeses	
Fermented meat products	
Oriental fermentations	
Fermenter-derived products	
Microbial proteins	
Food additives	

Table 2 Some important toxigenic species of filamentous fungi and related mycotoxins

Species	Toxin
<i>Aspergillus flavus</i>	Aflatoxins B ₁ , B ₂ ; cyclopiazonic acid
<i>A. parasiticus</i>	Aflatoxins B ₁ , B ₂ , G ₁ , G ₂
<i>A. ochraceus</i>	Ochratoxin A; Penicillic acid
<i>A. versicolor</i>	Sterigmatocystin, cyclopiazonic acid
<i>Penicillium verrucosum</i>	Ochratoxin A, citrinin
<i>P. purpurogenum</i>	Rubratoxins
<i>P. expansum</i>	Patulin, citrinin
<i>Fusarium sporotrichiodes</i>	T-2 toxin
<i>F. moniliforme</i>	Fumonisin B ₁
<i>F. graminearum</i>	Deoxynivalenol, nivalenol, zearalenone
<i>Alternaria alternata</i>	Tenuazonic acid
<i>Stachybotrys atra</i>	Satratoxins

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