GOOD PRACTICES FOR ANIMAL FEED AND LIVESTOCK 1

training manual

ON-FARM MYCOTOXIN CONTROL IN FOOD AND FEED GRAIN

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Preface

When we eat food that we have grown or bought we are unwittingly consuming many substances other than the basic nutrients which we assume make up the food item. The same applies to the feed we provide for our domesticated animals. Many foreign substances are added by man to improve quality and prevent biological decay, these include insecticides, colourants, minerals, vitamins and preservatives. However, there are many materials that have a damaging effect and are not added by man, but are the result of infections by other organisms. For example, foods can contain bacteria that grow naturally on produce and which may lead to food poisoning if ingested. Other harmful substances may be picked up as a result of environmental pollution, such as undesirable pesticides and heavy metals.

One group of undesirable substances are mycotoxins. For the most part, their presence is beyond the control of man and can lead to severe harmful effects in both humans and livestock, culminating, in some instances, in death. Mycotoxins can be produced throughout the growing cycle but predominately after the crop has matured in the field, through the harvest and storage periods. Their presence affects farmers, their families and livestock, as well as consumers in town and cities who may ingest contaminated produce.

This booklet is directed at the farm situation, providing information that can be used to avoid mycotoxin contamination before food leaves the farm. A separate booklet addresses issues related to transport, marketing and urban consumption. Although it is aimed at farm situations, the booklet is intended to be used by extension personnel, both government and non-government employees, in their efforts to advise and assist rural communities. It is not a treatise on mycotoxins and fungi but rather provides practical advice to combat the problem. It is expected that those using this booklet will have a basic knowledge of biology and of agriculture, both production and post-harvest.

The first sections describe what mycotoxins are, how they are produced and how to recognise signs of their presence. The booklet is particularly concerned with recognising the presence of fungal moulds and provides advice on how to prevent moulds from growing. The latter half of the booklet provides information to enable farmers to minimise the risk from mould contamination whilst the crop is growing, during harvesting, and then after harvest, i.e. when threshing/shelling, and storing the crop. It also examines actions to take when storing flour and feed, silage and hay.

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Glossary

Aflatoxicosis	(aflatoxicoses: plural) diseases resulting from consumption of aflatoxins
ATA	alimentary toxic aleukia, a mycotoxin disease in humans
DON	deoxynivalenol (a mycotoxin)
ELEM	equine leukoencephalomalacia, a disease of horses
Metabolite	a substance produced as a result of a chemical change occurring during a biological process, such as respiration, photosynthesis in plants, cell growth etc.
Mycotoxicosis	(mycotoxicoses: plural) diseases caused by ingestion of mycotoxins
OTA	ochratoxin A, a member of the ochratoxin mycotoxins
ppb	parts (of the active substance) per billion (parts of the target: body weight or grain weight)
PPE	porcine pulmonary oedema, a disease of pigs
T-2	a trichothecene mycotoxin

What are mycotoxins?

Mycotoxins are among the most potent causes of cancer. Ingestion through the diet can pose chronic health risks that may result in liver and kidney disease and a suppression of the immune system. Death may occur as a result of acute poisoning. In livestock, mycotoxins can also modify the nutrient value and therefore the metabolism of feed, and alter the hormonal balance.

Mycotoxins are chemicals produced by fungal moulds. These moulds grow during production, harvesting or storage of grains, pulses, nuts, roots and other crops. As the moulds develop, they produce different chemicals, some of which enable the fungal colonies to grow and multiply but others, including mycotoxins, appear to provide no obvious benefit to the moulds (some of these chemicals are actually beneficial to man as in the case of antibiotics, like penicillin). Mycotoxins are known as 'secondary metabolites' as they have no direct effect on the development of the mould, though they do give it an advantage over competing organisms, such as other moulds, yeasts and bacteria. These toxins, or poisons, disrupt cell structures and interfere with vital cellular processes, allowing the mould to compete successfully with other organisms. They also cause problems for humans and animals that ingest them. We will see just how dangerous these chemicals can be.

Not all moulds produce mycotoxins. Not even all strains of a species of mould that produces mycotoxins are able to do so. Nobody is aware of the precise factors that determine whether a growing mould will produce a mycotoxin or not. It is, therefore, not possible to anticipate whether a mould growing on a crop will release mycotoxin, but it is always better to assume that it will do so. It is better to be safe than sorry! By doing so, the chance of coming into contact with highly toxic substances can be reduced or avoided.

The most common mycotoxins known to cause health problems in man and livestock are:

- Aflatoxins
- Fumonisins
- Trichothecenes, especially T-2 toxin also known as vomitoxin
- Deoxynivelenol (DON)
- Ochratoxins, especially ochratoxin A (OTA)
- Ergot toxins
- Zearalenone

Some of the other toxins which may affect health are listed in Table 1.

SIGNS OF MYCOTOXINS AND MOULD GROWTH

Mycotoxins are not visible to the eye. Grain that shows no signs of mould infection may be contaminated with mycotoxin. These metabolites may be found on apparently uncontaminated grains that are in close proximity to others which show signs of mould growth. When a farmer sorts grain to be sold and removes the obviously discoloured, mouldy grain, even grain which shows no signs of mould growth may nonetheless be contaminated with mycotoxins, and thus find its way to the local market. However, this risk of contamination is relatively low, and for practical purposes, grain that does not show signs of mould infection can be regarded as being free of mycotoxin.

Moulds growing on grain and other commodities are generally clearly visible. They are often coloured and affect the appearance of the product on which they are growing. Produce infected with mould takes on an unappealing smell.

Physical signs of mould in animal feed include:

- Dustiness
- Caking
- Poor flow out of grain bins
- Feed refusal by animals for no apparent reason

FIGURE 1 Mouldy groundnut (a), cassava (b) and millet (c)

(a) (b) (c)

- 'Mouldy' and musty smell
- Darkening of feed and grain

The presence of mould has a major impact on the quality of grain. Nobody wants to eat mouldy food. Farmers will not allow mouldy grain to be used in the preparation of food for their families, unless there is a significant food shortage, such as when a famine occurs. Mould-contaminated grain is often discarded, is certainly difficult to sell, and will either cause grain to be rejected or at best, downgraded with a loss in value. However, mouldy grain is usually retained for brewing beer or is fed to livestock. We will see later why feeding poultry, pigs, goats, cattle and any other livestock with mouldy grain can seriously affect the health of the animals and be counterproductive.

FACTORS THAT AFFECT FUNGAL GROWTH

Fungal infection of food commodities can occur: during plant growth; once the plant has matured; during harvesting, drying and storage; during processing; and even when the processed product is waiting in the trader's store or on the consumer's shelf. Mycotoxin production, similarly, can occur at all these stages, from seed to shelf. Fungal growth and mycotoxin production are particularly influenced by moisture, temperature and oxygen content. Furthermore, grain and other food commodities that are damaged or stressed by drought, pests, and cultivation and harvesting practices, are more susceptible to mould growth.

Fungi multiply by releasing spherical spores. These germinate producing elongated projections known as hyphae. Hyphae multiply and branch and form a mass of fungal tissue, the mycelium, at which stage it is recognisable as mould.

FIGURE 2A Fungal life cycle *(Aspergillus)*

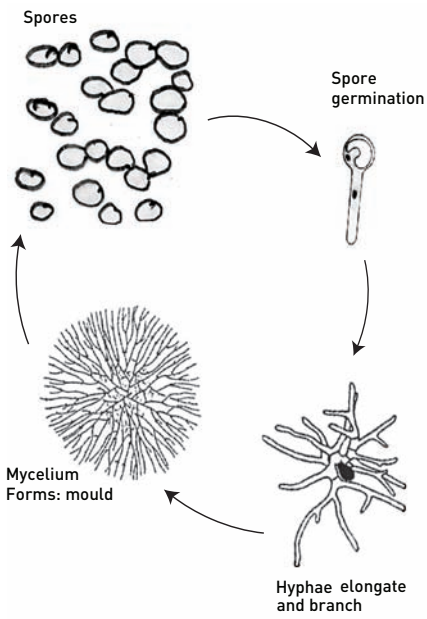
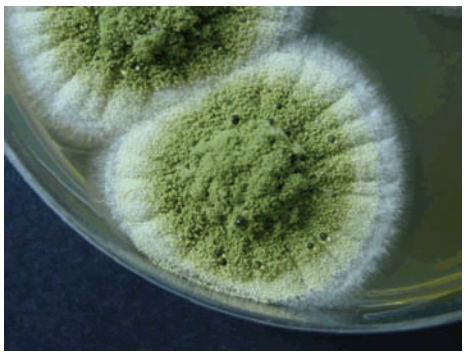


FIGURE 2B *A. flavus* mycelium (b)



Climate characteristics

Moulds require water for growth, reproduction and other biological processes. The amount of water in a plant or seed that is available to support these processes is known as its *'water activity'*. A minimum water activity of 0.70 will sustain growth of storage moulds, though for field moulds that produce mycotoxins water activity should be above 0.85. Water activity is similar to *moisture content* but is dependant on temperature and the chemical composition of the commodity. Thus the ambient temperature which affects the temperature of the grain and the type of commodity, whether it a cowpea grain, fresh tomato, groundnut pod or maize cob will affect how a mould will grow. For every 10°C, water activity rises 0.03.

The amount of moisture that is contained in air is known as the *humidity*. As air gets warmer it is capable of carrying more water; so humidity increases. The water in the air is in a state of balance with that contained in grains and other food products. When the air contains about 70% moisture, at a temperature of 27°C, most cereal grains will be able to hold 13-14% moisture; groundnuts will hold about 7% because of their high oil content. If the temperature then decreases the air will give up some of its water, which will be picked up by the grain. Conversely, if the temperature increases the air will take moisture from the grain and the grain moisture content will decrease. The relationship between water in the air and moisture content of grain is the relative humidity. As a very general rule and approximate guide *relative humidity* is about 100 times greater than water activity, so a relative humidity of 70% will equate to a water activity of maize of 0.70 when the temperature is about 25-30°C.

Most mycotoxin-producing fungi require the relative humidity to be above 70% in order for them to develop. So for wheat, maize, sorghum and millets stored in the tropics and sub-tropics a relative humidity of 70% will allow the grains to be kept safely without problems of mould contamination, as long as the moisture content of the grain itself is at 13-14% for cereals and 7% for groundnuts. If the moisture content for maize for example, is 20%, the grain will not be safe to store and moulds will grow.

Farmers cannot influence the moisture carried in the air or the ambient temperature. They may, however, influence the amount of moisture in their grain by drying it. They have to forcefully expel water and so upset the natural balance between the environment and their commodity. Where the climatic conditions are hot and humid, artificial drying may be the only way to reduce grain moisture content to prevent mould growth. In dryer climates this will be more easily achieved because the grain will naturally release water into the dry air until a balance or equilibrium is reached.

Other factors

Mycotoxin-producing moulds attack crops both before and after harvest. Pre-harvest fungal invasion is influenced by in-field damage caused by insects, birds, rodents, husbandry practices and adverse weather. Stress caused by drought, nutrient deficiency and untimely or excessive fertiliser application may also predispose towards fungal

establishment. Cracked grain may be readily invaded by air-borne, fungal spores. Maize cobs, heads of sorghum and millet, pods of legume crops such as groundnuts, beans and cowpeas that fall to the ground may be readily invaded by soil or detritus borne spores. Repeated planting of the same crop in the same or nearby fields favours fungal infection by increasing both the fungal innoculum and the insect population that attacks the growing crop.

Poor handling at harvest, during carriage from the field, whilst threshing and winnowing and during storage will similarly assist to encourage mould. Post-harvest pest attack, especially by insects, will make grain more susceptible to entry by fungal spores, and the production of water as a result of insect respiration will enable these spores to germinate and form mycelial clusters.

WHAT ARE THE EFFECTS OF MOULD ON ANIMAL FEED?

Mouldy feeds are less palatable and animals may eat less. This will lead to a reduction in nutrient intake and so decrease weight gain and milk production. Even if mycotoxins are not produced it has been estimated that the presence of mould alone will cause a loss in performance of 5-10%.

Mouldy feed may not be as digestible and the energy content will therefore be reduced. Furthermore, the mould itself will use the protein, carbohydrate and fat from the feed for its own development, reducing that available to the animal. Fat intake, especially, is reduced in mouldy feed and energy availability may be reduced by as much as 10%

Mould itself may cause health problems, especially respiratory disease. Breathing in mould spores for example, causes farmer's lung in man.

Numerous highly toxigenic species of Aspergillus, Penicillium and Fusarium have been found in damp hay and straw. As a result, a large range of toxins including patulin, aflatoxins and sterigmatocystin can be found in insufficiently dried hay and straw. In store, these moulds continue to be present for many months.

FIGURE 3 **Mould growing in a chicken's lung**

WHICH MOULDS PRODUCE MYCOTOXINS?

There are more than 300 known mycotoxins but we know very little about most of them. This manual focuses on the most common and important of these. The main mycotoxin producing species are shown in Table 1.

The genus *Fusarium*, which is a pathogen occurring during plant growth, produces more than 70 different toxins of which the most important are fumonisins, deoxynivalenol (DON), zearalenone and trichothecenes, which include T-2 toxin or vomitoxin, toxins that have only in recent years begun to be studied in depth. Some species of *Fusarium* can produce up to 17 different mycotoxins simultaneously.

Fusarium is responsible for head blight and scab in wheat and barley and ear rot in maize in cereal growing areas of Africa, Asia and North and South America. It can also be found on forage in the field. *Fusarium* species generally require a water activity of above 0.88 to develop so are rarely carried over in to stores after harvest, though they it may still develop on the standing crop if harvest occurs during periods of rainfall. However, although the mould may not,

itself, be carried over into the store the mycotoxins it produces may well be. *Fusarium verticillioides* and *F. pallidoroseum* predominate on cereals in the tropics and sub-tropics; *F. graminearium*, perhaps the most common of the genus, prefers humid-temperate conditions, which do occur in many parts of sub-Saharan Africa and Latin America. *Fusarium* toxins are not apparently carried over into meat, milk or eggs.

The most common, important and widely studied mycotoxins in tropical and semi-tropical areas are those of the aflatoxins complex, produced by *Aspergillus*, a post-harvest mould. There are several different aflatoxins; B1 is the most toxic. The other post-harvest genus of importance is *Penicillium*. Higher temperatures and humidities in the tropics mean that both these moulds have species that can develop on crops pre-harvest, once the grains have begun to dry, sometimes causing significant damage. For example, *A. flavus* and *A. niger* cause ear rots of maize and *A. flavus* can produce aflatoxins in the field. Ear rot infection and aflatoxin production starting in the field can carry over into store. *P. verrucosum* produces ochratoxin A (OTA) as does *A. ochraceus*.

Slow drying of grains and oilseeds after harvest can allow the continued growth of *A. flavus* and a rapid rise in aflatoxin concentration. If the commodity is improperly dried or allowed to become wet after drying a succession of fungi can then invade the grain dependant on the water activity. Species of *Eurotium* are the most common early invaders of improperly dried grain, leading to a loss of germination and quality. A succession of *Aspergillus* and *Penicillium* species then invade if the water activity is high enough. Species of fungi from these three genera are the most frequently isolated organisms from most stored durable crops.

Mould and mycotoxin problems are unlikely to occur if storage takes place mainly during dry periods of the year. Critical periods for contamination are: at the end of the growing season; when the crops are mature and begin to dry; during harvesting; and during the first months immediately after the harvest is brought to the farm. At maturity, when the plants begin to die back, continuous rain will slow the drying process and facilitate the development of *Fusarium* and production of its mycotoxins. Colonies of these fungi may well be carried over into the store and continue to grow if the relative humidity remains elevated. Prolongation of the rains during harvest – crops can remain in the field for many weeks before being removed for storage – similarly provides ideal conditions for many post-harvest fungi and particularly for *Aspergillus*. During the early months of storage, when grain may have a moisture content of 20% (water activity of 0.8+) or higher, fungal development will occur in the moist crop unless it is dried rapidly.

FIGURE 4 ***Aspergillus*** **ear rot (a) and** ***Fusarium*** **ear rot (b)**

(a)

(b)

TABLE 1 MAJOR MYCOTOXIN PRODUCING GENERA

Fungi	Mycotoxin	pre or post harvest	Climatic requirements	food commodities contaminated	COMMENT
Fusarium verticillioides (moniliforme), F. proliferatum	Fumonisin	Pre-harvest	Cosmopolitan	Wheat, maize and products, hay	Very stable, can survive cooking, soluble can be removed by wet milling and nixtamalisation
F. graminearium	Deoxynivalenol (DON or vomitoxin)	Pre-harvest	Cosmopolitan	Wheat, maize, millet, hay (zearalenone)	Very stable, can survive cooking, soluble can be removed by wet milling; not transferred to milk, meat or eggs
F. graminearium	Zearalenone				
F. graminearium, F. culmorum, F. poae	Trichothecenes	Pre-harvest	Temperate to cold	Wheat, maize (overwintered), hay	Very stable, can survive cooking
F. sporotrichioides	T-2 toxin				
Aspergillus ochraceus	Ochratoxin A	Post-harvest	Warm, humid	Cereals and cereal products, pulses, dried vine fruit, beverages, spices, groundnuts	Has been found in pig meat
Penicillium verrucosum	Ochratoxin A	Post-harvest	Temperate		
Penicillium sp., Aspergillus sp.	Citrinin				
Penicillium sp., Aspergillus sp.	Cyclopianzonic acid				
Aspergillus sp.	Sterigmatocystin				
Claviceps purpurea	Ergot	Pre-harvest	Mainly temperate	cereals	
Various	Patulin	Post-harvest		Mouldy fruit, vegetables, cereals, other foods.	Destroyed by fermentation
Aspergillus flavus, A. parasiticus	Aflatoxins (B1, B2, G1, G2, M)	Mostly post-harvest	Tropical and sub-tropical	Maize, wheat, groundnuts, other edible nuts, figs, spices, soya bean, cotton seed, oil palm kernels, copra, coconut oil, cassava	Moulds do not grow in silage but aflatoxins can survive ensiling process.

Fusarium toxins are apparently not carried over into milk, meat and eggs (M. E. Doyle (1997): http://www.wisc.edu/fri/fusarium.htm :5). Although fumonisins and DON are not the most toxic of the *Fusarium* toxins they are the most frequently detected and therefore the most associated with human and animal illness (this applies to US in particular and maybe also Europe, but may not be relevant for Africa).

Other mycotoxin producers including the growing crops *Claviceps purpurea* (ergot). This fungus produces toxic alkaloids from dark brown to black bodies (sclerotia or ergots) that stick out from the seed head of infected plants, commonly cereals and especially rye. Ergots are visible to the naked eye and look like rat droppings when removed from the seed head. They may grown up to 2.5 cm in length. Ingestion can result in 'ergotism', neurological disorders, including tremors, staggers, convulsions and necrosis, and gangrenous disorders such as sloughing of the hoof or

feet and tail as well as dry gangrene. A cessation of milk production in post-partum females is also common.

Another important genus is Stenocarpella (Diplodia), which causes stem and ear rots in maize. The composition of its mycotoxins are as yet unknown but the effects can be severe in livestock. Mycotoxins of *S. maydis* cause diplodiosis in cattle, symptoms of which are loss of co-ordination, nervous system defects, paralysis and death. In sheep, diplodiosis causes abortions, and in poultry reduced egg laying and growth. It is not known whether these moulds have any effect on humans.

Some other disease causing fungi include *Neotyphodium*, which causes dry gangrene, and *Rhyzoctonia*, which produces slaframin causing sialorrhea or excessive salivation in ruminants.

WHAT ARE THE SYMPTOMS OF MYCOTOXIN POISONING?

In livestock, aflatoxins B1 and M1, ochratoxin A (OTA) and sterigmatocystin have been found to be carcinogenic. In humans, aflatoxins and OTA have been classified as carcinogenic, and fumonisin B1 as a possible carcinogen. Patulin, a toxin produced by both *Aspergillus* and *Penicillium* species on fresh fruit, and which occurs particularly in fruit juices, is also a health hazard.

The effect of mycotoxins on animal production is influenced by the type and concentration of toxins present; the exposure period; the age and type of animal; and its nutrition and health status. Mycotoxins tend not to be recognised by the body's defence mechanism so that antibodies are not produced in response to their ingestion. Thus the effects of mycotoxin poisoning are often chronic and gradual, so making it frequently difficult to diagnose or monitor the effects before symptoms become severe. The situation is made worse because more than one mycotoxin may be present; for example aflatoxins and sterigmatocystin may be produced at the same time by *Aspergillus flavus.*

In man and animals mycotoxins target different organs. The following is a summary of which organs in the body and which physiological systems are attacked by different mycotoxins (it must not be assumed that ingestion of these toxins will always have the same effect as the dosage ingested will influence what the mycotoxin does).

• Digestive system including liver, mucous membranes – fumonisins, T-2 toxin, patulin and DON;

FIGURE 5 Ergot on millet

FIGURE 6 *Stenocarpella (Diplodia)* damage

- Respiratory system, lungs – trichothecenes, fumonisins;
- Nervous system – fumonisins, trichothecenes, cyclopianzoic acid and ergot;
- Cutaneous system, skin – trichothecenes;
- Urinary system, kidneys – fumonisins, ochra toxin A;
- Reproductive system, T-2 toxin, zearalenone;
- Immune system – aflatoxins and many others;
- Vascular system including blood vessels, liver –aflatoxins, sterigmatocystin.

Symptoms occurring in individual animals and man are described in more detail below.

Humans

Aflatoxins are at present the only mycotoxins that have caused human fatalities as a result of acute poisoning. Known fatalities were first recorded in India in 1974, when unseasonal rain and food scarcity forced people to eat maize that was heavily contaminated with this mycotoxin. Thereafter, contaminated maize consumption has resulted in human deaths in Kenya in 1982 and, more recently, in 2004 and 2005 when more than 200 people died.

Aflatoxins, like many of the other mycotoxins, suppress the immune system in humans and so increase the susceptibility to infection by other microbes. Aflatoxins have similar effects to those caused by HIV/AIDS and these toxins may even make people more readily susceptible to HIV/AIDS.

Aflatoxin B1 is a very potent human carcinogen, in particular causing liver cancer. Liver damage results in abnormal blood clotting, development of jaundice, haemorrhaging and a reduction in the immune response.

The trichothecene, T-2 was thought to be responsible for causing Alimentary Toxic Aleukia (ATA) in Siberia during WWII. Thousands of people were affected when they had to eat grain which had been allowed to over-winter in the fields. Symptoms included vomiting, acute inflammation of the digestive system, anaemia, circulatory failure and convulsions. ATA also occurred in Kashmir, India in 1987, where people ate bread made from mouldy flour; symptoms included vomiting, diarrhoea, inflammed throat and bloody stools. T-2 may be carcinogenic.

In combination with T-2 and other trichothecenes, DON resulted in human illness (mycotoxicosis) in India. Similar outbreaks occurred in Japan and China, the latter in 1984/5 from ingestion of mouldy maize and wheat, causing nausea, vomiting, abdominal pain, diarhhoea, dizziness and headache; the onset of symptoms occurred within 30 minutes. Human oesophageal cancer in Transkei, South Africa and China has been associated with presence of *F. verticillioides*, which produces fumonisins.

Cattle

Initially, the reduction in performance as a result of mycotoxin ingestion may not be recognisable. Within days or weeks the effects of continued mycotoxin consumption becomes more pronounced. Animals go off their feed, may suffer from diarrhoea, haemorrhaging, ketosis (excess sugar in the blood: diabetes) and from a displaced abomasum (4th stomach). Furthermore, reproductive effects, such as swollen vulvas and nipples, vaginal and rectal prolapse, may occur.

Mycotoxins can be detoxified in the rumen but cattle may still suffer the effects of the toxins. It is not possible to predict just what the effects are likely to be so it is always better to avoid feeding cattle mouldy feed if possible. Dairy cattle are more susceptible than beef cattle.

All livestock is affected by aflatoxins. Mature animals tend to be less susceptible than breeding and growing stock but young, pre-ruminant animals are the most at risk.

Feed levels of 60-100 ppb aflatoxins may effect performance. Chronic symptoms can occur with continued intake of 700-1000 ppb in 200-kg cattle and death has occurred within five days in feeding trials when the diet has contained 10,000 ppb.

BOX 1 SYMPTOMS OF AFLATOXICOSIS IN CATTLE

Acute effects

- reduced feed intake
- reduced weight gain or weight loss
- reduced feed efficiency
- increased susceptibility to stress
- decreased reproductive performance
- severe haemorrhaging resulting in death

Chronic effects:

- unthriftiness
- anorexia
- prolapse of the rectum
- liver and kidney damage, resulting in prolonged blood clotting time
- depression of the immune system
- oedema in the abdominal cavity and gall bladder

Even lower levels of aflatoxins may cause these effects when other mycotoxins are also present.

In dairy cattle, milk yield is reduced by aflatoxins and DON consumption, and both reduce reproductive efficiency. Aflatoxin M1 (a metabolite of aflatoxin B1) can be secreted into the milk at 1-2% of dietary intake when feed levels are 50 ppb or more. However, if contaminated feed is removed, milk residues disappear in 48-72 hours.

Zearalenone may give rise to abortions in dairy cattle, as well as reducing feed intake, milk production and cause vaginitis, vaginal secretions, poor reproduction performance and mammary gland enlargement in virgin heifers. T-2 causes feed refusal, gastroenteritis and may lead to death.

Poultry

Aflatoxins were responsible for the first major acute mycotoxin outbreak. In the United Kingdom in the early 1960s, aflatoxins caused 'Turkey X' disease, which was associated with the death of thousands of turkeys, ducklings and other domestic animals that had eaten a diet containing contaminated groundnut meal.

Less than 20 ppb aflatoxins in the diet will reduce resistance to disease, decrease ability to withstand stress and bruising, and make birds unthrifty and their condition generally poor. Young birds are more susceptible than laying hens, and ducks and turkeys are particularly at risk.

Zearalenone causes impaired fertility, stillbirths and reduced sperm quality. T-2 toxin and other trichothecenes may: suppress the immune system; reduce feed intake and cause weight loss; cause skin irritation (feather loss) and mouth lesions, diarrhoea, internal haemorrhaging and severe oedema of the body cavity. Death may well result.

Pigs

Nursing or nursery age swine are sensitive to aflatoxins. Feed levels of 100-400 ppb affect breeding stock, unweaned and growing pigs (less than 100 kg). As with other animals, there is usually a reduced ability to convert feed leading to a reduced rate of weight gain. Levels of 400-800 ppb have caused liver damage, bleeding disorders, suppression of the immune system, abortion and death.

BOX 2 EFFECTS OF AFLATOXICOSIS IN POULTRY

- stunted growth as a result of poor food conversion
- increased mortality
- reduced egg size
- reduced egg production
- liver and kidney disorders
- leg and bone problems
- suppression of the immune system with increased susceptibility to bacterial infections
- decreased blood clotting results in down grading and rejection due to massive bleeding and bruises

Zearalenone has similar effects as it does in poultry, causing swollen vulvas, and vaginal or rectal prolapse in pre-pubertal gilts. It also causes the uterus to enlarge and swell or become twisted, and the ovaries to shrink. In boars, the testes atrophy and mammary glands enlarge. Generally, there is a decrease in fertility.
DON can cause severe vomiting and may induce pigs to reject feed.

Fumonisins cause porcine pulmonary oedema (PPE), an accumulation of fluid in the lungs. OTA is associated with kidney damage.

Sheep and goats

The effects of aflatoxin ingestion are similar to those occurring in other ruminants. The liver and kidneys may be damaged, and the animals become anaemic. Early symptoms include depression, loss of appetite, weakness and slow movement.

Feed containing patulin cause nasal discharges, and loss of appetite and rumination, and body weight.

Horses

As non-ruminants horses may be more susceptible to mycotoxins, which may be responsible for:

- colic
- neurological disorders
- paralysis
- hypersensitivity
- organ deterioration
- reduced rate of growth
- poor feed efficiency
- impaired fertility
- death

The cumulative effects of feeding low levels of mycotoxins may result in a gradual deterioration of body organ function. This affects growth rate, feed efficiency, fertility, respiration rate, the ability to perform work and life span.

Working horses have a high energy requirement and require high concentrate intake, and would be most susceptible to problems with mycotoxin-contaminated grain. Lightly worked horses fed less grain are more likely to eat mycotoxin-contaminated hays or forages, such as cereal stover. Since mouldy forages are less palatable than normal forage, horse may well refuse feed before ingesting sufficient to do severe intestinal damage. Instead they may suffer mild colic. Moulds affecting feed grain do not usually affect palatability and so those horses fed grain are those most often exposed to mycotoxins.

Maximum aflatoxins levels for non-breeding, mature horses (2-yr and above) should be 50 ppb. Growing, breeding and working horses should receive aflatoxin-free diets. However, as the effects on horses are not well understood all horses should be fed diets free of mycotoxin.

Fumonisins, which seem to be particularly toxic to horses, cause ELEM (see Table 2), which is characterised by lesions in the brain. Incidences of ELEM have been reported in USA, Argentina, Brazil, Egypt, South Africa, China; it may well occur in other countries where insufficient expertise prevents diagnosis.

General effects of mycotoxins

A summary of the more specific effects of mycotoxins is shown in Table 2. Aflatoxins are carcinogenic, with the liver being especially susceptible. Trichothecenes, and T-2 in particular, are responsible for causing haemorrhaging in farm animals. Fumonisin B1 is a central nervous toxin, and also affects liver, pancreas, kidney and lung in many animals.

Aflatoxins and ochratoxins have been found in milk, meat and eggs. This is a particular concern for human health in developing countries where monitoring is not a routine or regular procedure, and where animals are likely to consume high levels of these toxins in feed.

Penicillium species are also responsible for producing two toxins, penitrem-A and roquefortine C, that induce tremors. These symptoms have been mostly observed in dogs that have

been allowed to roam around trash and composting mouldy food, especially dairy products. Low quantities of the toxins may cause tremors which last for several days but larger doses may cause seizures and result in death. Other animals that have been affected by these mycotoxins include cattle, sheep, rabbits, poultry and rodents.

It is clear that mycotoxins can have serious repercussions for the livestock industry. Even though our knowledge of the effects of many of the toxins is sketchy it is best to err on the safe side and prevent animals from ingesting mouldy grain and feed.

The following sections describe how we can avoid grain becoming contaminated with mould.

TABLE 2 SYMPTOMS OF MYCOTOXINS IN ANIMALS CONSUMING CONTAMINATED FEED

Mycotoxin	Fungi associated	Symptom/toxicology
Fumonisin	*Fusarium verticillioides (moniliforme), F. proliferatum*	Equine leukoencephalomalacia (ELEM), porcine pulmonary oedema, liver and kidney damage in other domestic animals, *(oesophagal cancer in humans: no proven causal link)*
Deoxynivalenol (DON or vomitoxin)	F. graminearium	Feed refusal, reduced weight gain, diarrhoea, vomiting, reduced reproductive performance, increased mortality, reduced egg or milk production, (nausea and headaches in humans)
Zearalenone	F. graminearium	Oestrogenic syndromes, mammary and vulvar swelling, uterine hypertrophy, infertility, increased blood clotting time, increased mortality, reduced growth, increased susceptibility to disease
Trichothecenes	*F. graminearium, F. culmorum, F. poae, , F. solani, F. nivale F. sporotrichioides, (T-2 toxin)*	Alimentary toxic aleukia (ATA), digestive disorders, reduced feed efficiency, reduced growth, bloody diarrhoea, reduced egg and milk productionnecrosis, haemorrhages, oral lesions in broiler chickens, increased blood clotting time, increased mortality.
Ochratoxins	*Penicillium verrucosum (temperate climates), Aspergillus ochraceus*	Porcine nephropathy, renal toxicity, immunosuppression, various symptoms in poultry
Citrinin	*Penicillium citrinum., Aspergillus sp.*	Kidney damage
Cyclopianzonic acid	*Penicillium cyclopium, P. commune, P. camembertii, Aspergillus flavus, A.versicolor*	Neurotoxin
Sterigmatocystin	*Aspergillus nidulans, A. versicolor*	Carcinogen, mutagen, affects liver
Ergot	*Claviceps purpurea*	Vertigo, staggers, convulsions, temporary posterior paralysis, and death. Decreased blood supply. Reduced growth, tail loss, reduced reproductive efficiency in pigs.
Patulin	Various	Depress immune system, carcinogen, neurotoxin, stomach irritant, nausea, vomiting, ulcers, haemorrhages
Aflatoxins	*Aspergillus flavus, A. parasiticus*	Liver necrosis, liver tumours. Reduced growth, depressed immune response, carcinogen;