Mycotoxin toxicity to animals

Disclaimer

Most inquiries about mycotoxins concern incidents of the poisoning of farm animals, often very expensive breeds. The toxicological data given in this section is only meant to be a guide to the relative toxicity of different compounds to different animals. It is mainly taken from Mycotoxins in Grain, by Miller and Trenholm, Eagan Press, St. Paul USA, 1994. Other publications may contradict the data in some instances.

In this article the concentrations have been given in parts per million (ppm) and parts per billion (ppb):

- 'n' = nano, 'u' = micro, 'm' = milli
- 1 ppm = 1 mg/kg = 1 ug/g
- 1 ppb = 1 ug/kg = 1 ng/g

Mycotoxins in grain

Mycotoxins are complex, stable chemicals which typically cause liver damage or damage to the immune system and may cause cancer. The presence of fungi and mycotoxins has affected the health of human and animal populations profoundly. Because mycotoxins are natural compounds, it is difficult to set acceptable intake levels for long term consumption, and very few limits have been set for mycotoxins in foods or feeds. The toxicity of naturally occurring mycotoxins is generally much higher than that of grain protectant insecticides. (The acceptable daily intake for ochratoxin is 16 ng/kg bodyweight, and that of fenitrothion is 5000 ng/kg bodyweight.) Limits for the most toxic compounds are set at the ppb (parts per billion) level. 1 ppb = 1 ng/g or 1 ug/kg.

Aflatoxin

Pre-harvest production of aflatoxin is a problem in maize, but in other cereals aflatoxin is only produced if there is fungal attack in storage. This can be prevented by keeping the commodity below the water activity requirement for growth of the Aspergillus spp. fungi. Aflatoxin in mouldy feed can come through in the milk of dairy animals. This is a serious health issue. Aflatoxin is very toxic to poultry, particularly ducklings, but is not so harmful to beef cattle (the table, Australian mycotoxin limits, lists the stockfeed limits for aflatoxin). The only international Codex standard (in draft) is a limit of 0.05 ppb of aflatoxin M1 in milk. Highly sensitive analysis by ultraviolet (UV) fluorescence, high performance liquid chromatography (HPLC) has a detection limit of 0.5 ppb. Confirmation can be achieved using thin layer chromatography (TLC), with a detection limit of 1 ppb. Many quick tests are available.

See Updates for a list of ELISA kits and suppliers.
Ochratoxin

Ochratoxin A (OA) commonly occurs in wheat and barley stored moist over winter in temperate climates. It rarely occurs in Australian conditions, but can occur in Australia when grain is stored in cool damp conditions (Webley et al.1997). OA occurs in animal products such as kidney, milk and bacon in Europe due to the feeding of contaminated grain to livestock. OA causes renal–hepatic toxicity, depletion of lymphoid cells, and reduction of bone marrow cells. The main guideline for allowable amounts of a contaminant in the diet is the acceptable daily intake (ADI) expressed as ug or mg of contaminant per kg of bodyweight (ug/kg-bw). These ADIs are set by the United Nations Food and Agriculture Organisation and the World Health Organisation through Joint Committees, particularly the Codex Committee for Food Additives and Contaminants. The Codex Committee has established a provisional tolerable daily intake for OA of 16 ng per kg bodyweight (44th Joint Evaluating Committee on Food Additives and Contaminants or JECFA). This is about 1 ug per day, based on 60 kg bodyweight. Several countries have set limits from 5 to 50 ppb (ug/kg) in animal products for human consumption. JECFA has discussed a proposed 5 ppb limit in cereals—a limit which could have important trade implications.

OA is very toxic to poultry and results in decreased weight gain, reduced feed consumption, reduced egg laying, impaired kidney function at levels of 2 ppm. Other reports suggest effects occur at 1 ppm or less. Chickens are more sensitive to OA than to aflatoxins with LD 50s of 2-4 mg/kg-bw. OA causes nephropathy in pigs at less than 2 ppm in the diet, and the level that produces long term kidney damage may be lower than 1 ppm. Ruminants are tolerant of OA at levels greater than 100 ppm. ELISA test kits are sensitive to about 2 ppb. HPLC with UV fluorescence detection gives a minimum detection level of 2 ppb. TLC can also used for confirmation with a detection limit of 20 ppb.

DON

The mycotoxin deoxynivalenol (DON) occurs in wheat and other cereals when the growing conditions allow infection with the fungus Fusarium graminearum. This fungus produces the diseases headscab and crown rot. DON is the most frequently found toxin of a group called the trichothecenes. Compared with aflatoxin or OA, DON is not acutely toxic, but it has adverse effects on the immune system and is detrimental to animal welfare. The main symptom in pigs is feed refusal and vomiting, hence the popular name, vomitoxin. Reduction of immunity is a common effect of the trichothecenes.

DON is a problem in North America where the permitted guideline limit for DON on grain going into animal feeds is 10 ppm. In Europe, DON is rarer than in North America, but a related toxin, nivalenol (NIV), is found there. DON and NIV have been detected frequently in cereals in China, Korea, Japan, Russia and elsewhere. Extensive surveys by Food Science Australia and AWB Ltd have shown that Australian wheat is generally free from DON and almost free from Fusarium graminearum.

In pigs, low levels (above 1 ppm) of DON may cause feed rejection, and about 5 ppm may produce vomiting. Cattle are tolerant of greater than 6 ppm DON over a long period and lambs can take more than 16 ppm DON. There is very little effect on poultry at levels of 10 ppm or greater. NIV is about 10 times more toxic than DON.
HPLC is used for deoxynivalenol, and nivalenol analysis. Commercial ELISA kits for deoxynivalenol, sensitive to about 250 ppb, are useful for routine screening.

**Fumonisin B1 (FB 1)**

Fumonisins are toxins which are produced in maize by the fungi *Fusarium moniliforme* and *F. proliferatum*, and were discovered only in 1988. Fumonisins are unusual in producing different effects on different animals and are particularly toxic to horses, causing equine leucoencephalomalacia (ELEM) which produces holes or lesions in the brain. Toxicological problems can develop in horses at 20 ppm or less. 35 ppm or more will produce ELEM in horses. Fumonisins cause porcine pulmonary edema (PPE) in pigs, a disease in which the lungs fill with fluid. About 50 ppm FB 1 in the diet produces liver dysfunction in pigs and 200 ppm produces severe PPE. FB 1 is a severe problem in poultry production as it causes immune system deficiency. 100 ppm causes liver lesions and reduced performance in poultry. Compared with pigs and horses, cattle are relatively tolerant of FB 1, with no effect from 150 ppm in the diet for 31 days. Other effects are nephrosis in sheep, and esophageal cancer in humans. Fumonisin contamination is widespread in the USA, South Africa, China and Europe. It is also present in Australia.(Bryden 1995)

**Zearelenone (ZEN)**

The animals most sensitive to ZEN are pigs. Reproductive dysfunction occurs at about 1 to 3 ppm. In cattle, abortions and fertility disturbances occur above 10 ppm ZEN. Sheep are more affected by ZEN than cattle are. Poultry are not affected by ZEN even at high concentrations. Zearalenone strongly fluoresces under UV and is easily detected in HPLC with a detection limit of 5 ppb. Commercial ELISA kits have a sensitivity of about 5 ppb.

**T-2 toxin**

T-2 toxin causes reduced feed uptake, lower weight gain, necrosis of alimentary tissues, neural disorders and abnormal feathering. Toxic effects may appear at a level of about 5 ppm T-2 in the diet, although this is unlikely to be fatal. T-2 may produce haemorrhagic disease in cattle, causing reduced feed uptake, loss of hair, decreased milk yield and multiple haemorrhage. 2 ppm of T-2 toxin in maize has been said to cause death in dairy cattle, but other reports suggest that 20 ppm of pure T-2 toxin can be tolerated. Analysis of T-2 toxin and HT-2 is by GC-ECD. The presence of T-2 toxin can be confirmed by ELISA.

**Alternaria mycotoxins**

Australian grain and oilseeds are often heavily infected with *Alternaria* fungi and may contain mycotoxins produced by this group of fungi, including tenuazonic acid (TeA), alternariol (AOH), and alternariol monomethyl ether (AME). These are generally less toxic than other commonly occurring mycotoxins.

TeA is toxic to chickens. The LD50 for chickens by single dose esophageal intubation has been given as 37 mg/kg/bw. AOH and AME appear to be less toxic, no symptoms being observed in rats or chicks when AOH and AME were fed for 21 days at 39 and 24 ppm respectively. In Queensland, broilers fed on mouldy sorghum containing 7-10 ppm AOH and 3-7 ppm AME showed 15% decreased body weight, poor feed conversion and enlarged liver and pancreas.
AOH, AME, and altenuene are measured by HPLC with UV fluorescence. TeA is
determined by HPLC with UV absorption. AOH and AME can be confirmed by TLC
with UV fluorescence to a minimum sensitivity of 30 ppb but TeA fluoresces only
after spraying the plate with terbium chloride, and this is not sufficiently
sensitive to confirm the presence of the low levels of TeA (less than 150 ppb)
identified by the HPLC method. The levels can be confirmed by PDA detection.
There are no commercial ELISA kits at present.

**Phomopsin**

*Phomopsis leptostromiformis* is a pathogen, largely confined to lupins, which
produces several mycotoxins of which the principal one is phomopsin A. In years
of high rainfall at the time of seed maturity there may be 3 to 10 % of discolored
infected seed within the crop. The level of phomopsin in discolored seed may
reach 3.4 mg/kg (ppm). The disease lupinosis in sheep is caused by grazing on
lupin stubbles and manifests itself as a liver disease with jaundice and general
non-productivity. The oral LD 50 of phomopsin A in sheep is 108 µg/kg-bw. The
no-effect level is above 24 µg/kg-bw daily. Pigs are much less susceptible than
sheep. Phomopsin A produces liver cancer in rats.

Australia has a 5 ppb limit for human consumption.

**Corynetoxin**

Annual ryegrass toxicity (ARGT) causes the deaths of about 30,000 sheep per
year. In 1991, the estimated number of sheep poisoned in Western Australia was
over 88,000. The deaths are due to the presence of corynetoxin which is
produced by a bacterium, *Clavibacter toxicum*, which in turn invades galls caused
by the nematode *Anguina funesta* in ryegrass pastures. The PMTDI is 10-70
ng/kg-bw, similar to ochratoxin, and the oral lethal dose is about 3 mg/kg-bw.
The fodder industry has accepted a standard of 5 µg/kg, which is equivalent to
about 1 gall per kg of grain.

**Mycotoxins in pulses**

Generally grain legumes (pulses) do not support the growth of toxigenic fungi
and the production of mycotoxins. Aflatoxin and other toxins have been reported
in various types of legumes due to post harvest damage, but much less often
than in cereal grains.

**Mycotoxins in oilseeds**

Several *Alternaria* species can occur on oilseeds and produce toxins such as
tenuazonic acid which is toxic to birds. When selling oilseeds directly for
specialist animal feed, the seeds should be checked for *Alternaria* toxins.

**Mycotoxin in feeds**

Farm animals are more at risk from mycotoxin poisoning than are humans. This
is because a large proportion of their diet may come from one batch of feed, and
also because they may be fed peelings, screenings and other external parts
where the highest level of toxins occur. Whilst most incidents of mycotoxin
poisoning occur in farm animals, toxins present in mouldy feed can enter the
human food chain through meat or dairy products. This is particularly true of
aflatoxin in milk and ochratoxin in bacon, and in kidney and other tissues, thus making both toxins a matter for major international health concern (see table, International mycotoxin limits).

Click to see the Mycotoxin sub-menu.