Introduction
Mycotoxins - toxic fungal secondary metabolites – are emerging as a significant factor in animal health and performance. Economic losses are due to effects on livestock productivity and direct losses in crop yield and stored agricultural products. A wide variety of mycotoxins are present in our environment and veterinarians are likely to be required to diagnose and treat such problems on a larger scale as diagnostic procedures and available data become more plentiful and cost effective respectively. Fink-Gremels states that ruminants are generally considered to be less susceptible to the adverse effects caused by contamination of feeds with mycotoxins based on the assumption that the rumen flora degrade and inactivate mycotoxins, thus protecting the animal. A number of mycotoxins, however, resist rumen degradation, and may cause clinical signs of toxicosis. Moreover, due to their complex diet, cattle may be exposed to a varying number of mycotoxins, originating from different feed materials such as roughage and concentrates. Exposure to these complex mixtures of mycotoxins may result in unexpected health risks. This article, to a large extent, summarizes the work of Harris and Staples as well as various authors at the Mycotoxin Workshop May 22 – 24, 2013 in Ghent, and focusses largely on mycotoxins in ruminants.

Symptoms
Symptoms are often non-specific, may be wide-ranging and result from a progression of effects or of opportunistic diseases which may make a diagnosis difficult or impossible because of the complex clinical results. Symptoms vary depending on the mycotoxins involved and their interactions with other stress factors. The more stressed cows, such as freshly calved cows, are most affected, perhaps because their immune systems are already suppressed. Symptoms may include reduced production, reduced feed consumption, intermittent diarrhoea (sometimes with bloody or dark manure), reduced feed intake, unthriftiness, rough hair coat, reduced reproductive performance including irregular estrous cycles, embryonic mortalities and decreased conception
There generally is an increase in incidence of disease, such as displaced abomasum, ketosis, retained placenta, metritis, mastitis, and fatty livers. Cows do not respond well to veterinary therapy.

**Diagnosis**

Commercial laboratories provide screens for a number of mycotoxins but cost of analyses has been a constraint. Analytical techniques for mycotoxins are improving, costs are decreasing, and several commercial laboratories are available that provide screens for an array of mycotoxins. Collection of representative feed samples is a problem because molds can produce large amounts of mycotoxins in small areas, making the mycotoxin concentrations highly variable within the lot of feed. As mycotoxins can form in the collected sample, samples should be preserved and delivered to the lab quickly. Samples can be dried, frozen, or treated with a mold inhibitor before shipping. Collected samples are finely ground and subsampled for analysis; this step is the second-largest source of error in an analysis. Subsamples are extracted and the extract/s purified using one of several techniques, and then the mycotoxins are measured. Toxin determination may be by thin-layer chromatography plates, high-performance liquid chromatography, gas-liquid chromatography, enzyme-linked immunosorbent assays, and spectrophotometer, or by other techniques.

The development of liquid chromatography-mass spectrometry methods for the simultaneous detection and quantification of a broad spectrum of mycotoxins has facilitated the screening of a larger number of samples for contamination with a wide array of less well-known mycotoxins and other metabolites. Streit et al demonstrated up to 69 fungal metabolites in feed and feed raw materials and the total number of detected metabolites amounted to 139. They report that *Fusarium* mycotoxins were most common, but a number of *Alternaria* toxins also occurred very often. In addition masked mycotoxins (i.e., mycotoxin conjugates), namely deoxynivalenol-3-glucoside (75% positives) and zearalenone-4-sulfate (49% positives), were also frequently detected. Their study highlights the shortage of knowledge in estimating toxicological potential and possible synergistic effects of multiple toxins.

Mold spore counts may not be very useful, but their presence is a gross indication of the potential for toxicity. Mold identification can be useful to suggest which mycotoxins may be present. Because tests for some potentially important mycotoxins such as PR toxin are not generally available, it is currently recommended to analyze silages for mold spore count and mold identification to provide some insight to possible problems. Blacklighting for bright-greenish-yellow fluorescence (BGYF) is often used as a screening technique for aflatoxin in corn grain, but it is very inaccurate. As far as we are aware, blacklighting is completely inappropriate for other mycotoxins.

Generally, laboratories provide analysis for only a limited number of mycotoxins, perhaps including aflatoxin, ochratoxin, DON, ZEA, fumonisin and T-2 toxin. Minimum detection levels may be limited because the purpose of the laboratory is often directed at finding high levels that cause acute toxicity and serious animal disease rather than low levels associated with chronic effects such as production losses, impaired immunity, and significant economic losses.

**Mycotoxins**
Aflatoxins

Aspergillus flavus and A. parasiticus produce the extremely toxic, mutagenic, and carcinogenic aflatoxins. Harris and Staples suggest that aflatoxins grow primarily on maize and Aspergillus flavus and Aspergillus parasiticus produce four major toxins: B1, B2, G1 and G2. These were named according to their fluorescence properties under shortwave ultraviolet light on thin-layer chromatographic plates; B1 and B2 fluoresce blue, whereas G1 and G2 fluoresce green. Metabolites of toxicological significance include: aflatoxin B1 (AFB 1) AFB 2 and AFM 1.

Harris and Staples report that Aspergillus species are capable of colonizing most of the important grain crops including maize, small grains, peanuts, cottonseed and most nut crops in warm and humid climates and during the stages of growth, harvest and transportation. Colonization of soybeans and small grains generally occurs in storage. Damage to the seed coat (pericarp) induced by insects, drought, hail, frost, or mechanical harvesting favours fungal invasion. Moreover, insects can also serve as carriers of fungal spores. Aflatoxin B1 is excreted in milk in the form of aflatoxin M1 and is considered a human health hazard.

Symptoms of acute aflatoxicosis in mammals include inappetence, lethargy, ataxia, rough hair coat, and pale, enlarged fatty livers. Symptoms of chronic aflatoxin exposure include reduced feed efficiency and milk production, jaundice, and decreased appetite. Aflatoxin lowers resistance to diseases and interferes with vaccine-induced immunity while in beef cattle an effect on weight gain and intake with diets containing 700 ppb aflatoxin has been reported.

Tremorgenic Neurotoxicosis

Aspergillus clavatus

Dr Matthew Legg of Bapsfontein Veterinary Clinic reported on a case of mycotoxicosis involving Aspergillus clavatus during December 2013. The fungus had grown on barley sprouts and he described symptoms as follows: clinically the affected animals had no fever, heart rate was normal and mucus membranes were pink. Neurologically they showed knuckling over (some severely) in the hind limbs and hyperesthesia. Some animals collapsed and showed small seizures with paddling, and then got up a few moments later. They had muscle fasciculations in the hind limbs and a few in the neck as well. Mortality rate was low. RA McKenie et al reported a similar finding in hydroponically produced sprouted barley in Australia. Affected animals developed posterior ataxia with knuckling of fetlocks, muscular tremors and recumbency, but maintained their appetite. A few animals showed reduced milk production, hyperaesthesia, drooling, hypermetria of hind limbs or muscle spasms. Degeneration of large neurones was seen in the brain stem and spinal cord grey matter.

Deoxynivalenol (DON)

Fusarium molds produce a mycotoxin known as deoxynivalenol (DON) which is commonly detected in feed and is sometimes called vomitoxin because it was associated with vomiting in pigs. The impact of DON on dairy cattle is not established, but clinical data has shown an association between DON and poor performance in dairy herds. Dairy cattle consuming diets contaminated primarily with DON (2.5 ppm) have responded favourably to the dietary inclusion of a mycotoxin binder, providing circumstantial evidence that DON may reduce milk production...
Dairy cows in the East Rand have been reported to have been affected by mycotoxins during the winter of 2014 (personal communication Dr Danie Nolte, Delmas). Dr Nolte reported dramatic reductions in milk yield (approximately 8 litres per day) and reduced feed intakes. Incidences of bloat, loose dung and undigested roughage in dung were noted with occasional downer cows and deaths showing primarily rumen stasis and ascites. A generalized loss of body condition in affected herds was noted. Lower grades of maize had been used initially in rations and were associated with the onset clinical symptoms described above and analyses of feed constituents revealed levels of DON ranging from 0.410 ppm in a TMR, up to 3 ppm in silage and 1.73 ppm in dairy meal. He further reported that when the maize constituents were replaced with better grades there was a dramatic improvement in all of the noted problems. This is consistent with a number of literature reports including those above.

DON has been associated with altered rumen fermentation and reduced flow of utilizable protein to the duodenum. Beef cattle and sheep have tolerated up to 21 ppm of dietary DON without clinical effects but pure DON added to diets appears not to have as much toxicity as does DON supplied from naturally contaminated feeds, possibly due to the presence of multiple mycotoxins in naturally contaminated feeds, which may interact to produce symptoms that are different or more severe than expected. For example, it is now known that fusaric acid interacts with DON to cause the vomiting effects, which earlier was attributed to DON alone.

According to Merck Veterinary Manual, DON is classified as one of the trichothecene mycotoxin group. On the basis of molecular structure, the trichothecenes are classed as non-macro cyclic (eg DON, T-2 toxin, diacetoxyscirpenol and others) or macrocyclic (satratoxin, roridin, verrucarin).

**Trichothecenes**

Toxicity of the trichothecenes, except for DON, is based on direct cytotoxicity and is often referred to as a radiomimetic effect (eg, bone marrow hypoplasia, gastroenteritis, diarrhoea, hemorrhages). The cutaneous cytotoxicity that follows on administration of these compounds is a non-specific, acute, necrotizing process with minimal inflammation of both the epidermis and dermis. Stomatitis, hyperkeratosis with ulceration of the esophageal portion of the gastric mucosa, and necrosis of the GI tract have been seen after ingestion of trichothecenes.

Sublethal toxic doses of the trichothecenes are highly immunosuppressive in mammals; however, longterm feeding of high levels of T-2 toxin does not seem to activate latent viral or bacterial infections. Hemorrhagic diathesis may occur after thrombocytopenia or defective intrinsic or extrinsic coagulation pathways. It appears that hemorrhage results from depression of clotting factors, thrombocytopenia, inhibition of platelet function, or possibly a combination of these. Refusal to consume contaminated feedstuff is a typical sign, which limits development of other signs. If no other food is offered, animals may eat reluctantly.

*Fusarium* molds also produce the very potent T-2 toxin. In dairy cattle, T-2 toxin has been associated with feed refusal, low feed consumption, decreased milk production, absence of
estrous cycles, gastroenteritis, intestinal hemorrhages and death. Dietary T-2 toxin at 640 ppb for 20 days resulted in bloody feces, enteritis, abomasal and ruminal ulcers, and death. Serum immunoglobulins and complement proteins were lowered in calves receiving T-2 toxin and a reduction in white blood cell and neutrophil counts has also been demonstrated. McLaughlin et al (1977) demonstrated that the primary basis of T-2 toxin reduced immunity is reduced protein synthesis.

Zearalenone (ZEA)
Zearalenone is a Fusarium-produced mycotoxin that has a chemical structure similar to estrogen and can produce an estrogenic response in animals. Zearalenone is associated with ear and stalk rots in corn and with scab in wheat. Controlled studies with ZEA at high levels have failed to reproduce the degree of toxicity that has been associated with ZEA-contaminated feeds in field observations. Studies in cows and heifers have resulted in smaller corpora lutea and reduced conception rates by about 25%. Case reports have described estrogenic responses in ruminants including abortions, vaginitis, vaginal secretions, poor reproductive performance, and mammary gland enlargement. In a field study (Coppock et al., 1990), diets with about 660 ppb ZEA and 440 ppb DON resulted in poor consumption, depressed milk production, diarrhoea, increased reproductive tract infections, and total reproductive failure.

Fumonisins: F. verticillioides
F. verticillioides produces fumonisin B1 which is associated with leukoencephalomalacia in horses, pulmonary edema in pigs, and hepatotoxicity in rats and it is thought to be a promoter of esophageal cancer in humans. Fumonisins are structurally similar to sphingosine, a component of sphingolipids, which are in high concentrations in certain nerve tissues such as myelin. Fumonisin toxicity results from blockage of sphingolipid biosynthesis and thus degeneration of tissues rich in sphingolipids. While fumonisin B1 is much less potent in ruminants than in pigs, it has now been shown toxic to sheep, goats, beef cattle, and dairy cattle. With high feeding levels, there were mild liver lesions and lymphocyte blastogenesis with elevated enzymes indicative of liver damage. Dairy cattle (Holsteins and Jerseys) fed diets containing 100 ppm fumonisin for approximately seven days prior to calving and for 70 days thereafter demonstrated lower milk production (6 kg/cow/day), explained primarily by reduced feed consumption (Diaz et al., 2000). Increases in serum enzyme concentrations suggested mild liver disease. Because of greater production stress, dairy cattle may be more sensitive to fumonisin than are beef cattle. Fumonisin carryover from feed to milk is thought to be negligible.

Diplodiosis: Diplodea maydis
Christo Botha 2002 reported on Diplodea maydis (new name Stenocarpella maydis)
He described this as one of the most prevalent moulds on harvested maize throughout the world causing ear rot. In the South African context, maize is produced mainly in the North-West, the north-western, northern and eastern Free State, the Mpumalanga Highveld and the Kwazulu-Natal midlands causing a neurotoxicosis of cattle and occasionally sheep on harvested maize fields in winter. It is a phytopathogenic fungus that causes stalk rot and cob rot. The mycelium is conspicuously coarse, white to grey resembling toothpaste. The mould and the kernels eventually turn a greyish brown colour and rot the entire ear. A distinguishing characteristic of ear rot is the appearance of raised black bumps on the moldy husk or kernels. These are the picnidia of the
fungus, where new spores are produced. Infections first appear at the base of the ear frequently penetrating through areas of insect damage. The fungus overwinters as mycelium, spores, and picnidia on corn residue or seed and favoured by cool, wet weather during grain fill. Spores are spread by splashing rain. The toxic principle is unknown but it affects the central nervous system in cattle leading to salivation, wide-base stance, tremors, ataxia, incoordination, stiffness, high-stepping gait and paresis/paralysis. Post mortem lesions are non-specific.

**Ergotism:**  *Claviceps spp.*
Several species of *Claviceps* may infect plants and produce toxins in fungal bodies called sclerotia or ergots, which are small black bodies similar in size to the grain. Ergotism primarily causes a gangrenous or nervous condition in animals. Symptoms are directly related to dietary concentrations and include reduced weight gains, lameness, lower milk production, agalactia, and immune suppression. Sclerotia concentrations above 0.3% are related to reproductive disorders.

**Fescue toxicity:**  *Neotyphodium or Epichloe spp.*
Fescue infected with *Neotyphodium or Epichloe* may contain toxic alkaloids and intake is associated with “fescue toxicity” characterized by lower weight gains, rough hair coat, increased body temperature, agalactia, reduced conception and gangrenous necrosis of the extremities such as the feet, tail, and ears.

**Ochratoxin:**  *Penicillium and Aspergillus spp.*
*Penicillium* and *Aspergillus* produce Ochratoxin A (OTA) and is a causative agent of kidney disease in pigs that has been referred to as mycotoxin porcine nephropathy (Krogh, 1979). The primary toxic effect is inhibition of protein synthesis (Creppy et al, 1984). In cattle, OTA is rapidly degraded in the rumen and thus thought to be of little consequence unless consumed by young pre-ruminant calves (Sreemannarayana et al, 1988). With high-grain diets, less of the dietary ochratoxin may be degraded in the rumen and thus be more toxic in those situations (Hohler et al, 1999). Moldy alfalfa hay containing *A. ochraceus* was implicated as producing OTA associated with abortions in cattle (Still et al, 1971). OTA in moldy forage has also been implicated in cattle deaths (Vough and Glick, 1993).

**PR Toxin:**  *P. roqueforti*
PR toxin, produced by *P. roqueforti*, has been suggested as the causative agent associated with moldy corn silage and unspecified herd health problems. *Penicillium* molds grow at a low pH and in cool, damp conditions and has been found to be a major contaminant of silage

**Patulin**
Patulin is produced by *Penicillium, Aspergillus* and *Byssochlamy* and in moldy fruits such as apples (most commonly) but may also be found in grains, especially wet grains, and silage. Patulin is an antibiotic against gram-positive bacteria. Added to rumen cultures at 0, 20, 40, or 80 mg per day, patulin reduced VFA production, fiber digestion, and bacterial yield (Tapia et al., 2005). The potential for patulin toxicity of livestock is thought to be low, but there are reported case studies of toxicity (Sabater-Vilar et al., 2004).
Citrinin
Citrinin can co-occur with OTA and is produced by both *Penicillium* and *Aspergillus*. Citrinin is a parasympathomimetic agent, and causes necrosis of tubular epithelial cells in the kidney, and in some cases, hepatotoxicity (Hanika and Carlton, 1994).

Other toxins
Many other mycotoxins may affect ruminants, but there is less information about them, or they are of less consequence. There is much less information available about mycotoxins in forages. The array of mycotoxins found in forages may be different from those found in grains and are of major importance in mycotoxicoses of ruminants. The most prevalent genera isolated from forage may be *Aspergillus sp* and *Penicillium sp* followed by *Fusarium sp* and *Gibberella sp*. Grass and maize silage may yield *Penicillium* followed by *Mucoraceae sp*, *Monascus sp* and *Aspergillus sp*.

Prevention and treatment
Prevention of mycotoxin formation is essential since there are few ways to completely overcome problems once mycotoxins are present. Drought and insect damage are most important in instigating mold growth and mycotoxin formation in the field. Soil contamination, delayed harvest, late season rain and cool periods may increase mycotoxins.

Grain storage and harvesting equipment should be maintained to avoid kernel damage and grains should not be allowed to remain at moisture levels greater than 15 to 18%. While there is little mold growth in grain below 15% moisture, drying to levels below 14% and preferably to <13% help to compensate for non-uniform moisture concentrations throughout the grain mass. High temperatures increase the amount of free moisture (water activity) in the grain which is the primary cause of mold growth in storage. Mycotoxin concentrations are greatest in the fines and cleaning can greatly reduce mycotoxin concentrations in the grain.

After harvest, prevention of mycotoxins in silage includes following accepted silage-making practices aimed at preventing deterioration primarily by quickly reducing pH and eliminating the oxygen. Accepted silage-making practices emphasize:

• harvesting at the proper moisture content,
• chopping uniformly at the proper length,
• filling the silo rapidly
• packing the silage sufficiently to exclude air,
• using an effective fermentation aid, and
• covering completely and well.

Infiltration of air after ensiling can allow growth of acid tolerant microorganisms, an increase in the pH, and then mold growth. *Penicillium* molds are acid tolerant and may grow if any air is present. Ammonia, propionic acid, sorbic acid, and microbial or enzymatic silage additives are shown to be at least partially effective at inhibiting mold growth by providing some of the acidity needed for preservation without sole reliance on microbial produced acids. Managing the feeding face of silos should ensure a clean cut and as little as possible disturbance of the silage mass.
Increasing dietary levels of nutrients such as protein, energy, and antioxidants may be advisable. Animals exposed to aflatoxin show marginal responses to increased protein. In some situations, poultry respond to water-soluble vitamins or to specific minerals, but data are lacking for cattle. Acidic diets seem to exacerbate effects of mycotoxins, and therefore adequate dietary fiber and buffers are recommended. Because mycotoxins reduce feed consumption, feeding management to encourage intake can be helpful. Dry cows, spring heifers, and calves should receive the cleanest feed possible. Transition rations can reduce stress in fresh cows and reduce effects of mycotoxins. Strategic use of mold inhibitors can be beneficial.

**Mycotoxin Binders**

The addition of mycotoxin binders to contaminated diets has been considered the most promising dietary approach to reduce effects of mycotoxins. The theory is that the binder decontaminates mycotoxins in the feed by binding them strongly enough to prevent toxic interactions with the consuming animal and to prevent mycotoxin absorption across the digestive tract. Potential absorbent materials include activated carbon, aluminosilicates (clay, bentonite, montmorillonite, zeolite, phyllosilicates, etc.), complex indigestible carbohydrates (cellulose, polysaccharides in the cell walls of yeast and bacteria such as glucomannans, peptidoglycans, and others), and synthetic polymers such as cholestryamine and polyvinylpyrrolidone and derivatives.

Research with mycotoxin binders has been ongoing for a number of years, and yet products are still not approved for the marketplace, and information for producers is limited. Due to the variations in manifestations and synergies between toxins gathering accurate evidence based definitive information is complex, expensive, time consuming, and solutions have not been forthcoming to any extent.

Activated carbon is a general adsorptive material with a large surface area and excellent adsorptive capacity. It has been recommended as a general toxin adsorbing agent and is routinely recommended for various digestive toxicities (The Merck Veterinary Manual, Eighth Edition, Merck & Co. Inc., Whitehouse Station, NJ). In certain studies the effects of activated charcoal have been variable and responses to charcoal did not exceed that seen with a clay-based binder (a hydrated sodium calcium aluminosilicate or HSCAS).

Generally it is suggested that charcoal may not be as effective in binding aflatoxin as clay-based binders but activated charcoal may be important in binding zearalenone and/or deoxynivalenol although it appears that binders may not necessarily be effective for deoxynivalenol.

Silicates are divided into subclasses, not by their chemistries, but by their structures. The silicate subclasses include neosilicates (single tetrahedrons), sorosilicates (double tetrahedrons), inosilicates (single and double chains), cyclosilicates (rings), phyllosilicates (sheets), and tectocilicates (frameworks). Silicates investigated as adsorbent materials are classified primarily as phyllosilicates and tectosilicates. It is suggested that this specific silicate mineral can bind with aflatoxin by chelating the β-dicarbonyl moiety in aflatoxin with uncoordinated metal ions in the clay materials.
The clay group is a subcategory of the phyllosilicates. Bentonite is a general clay material originating from volcanic ash and containing primarily montmorillonite as the main constituent. Montmorillonite clay is a hydrated sodium calcium aluminum magnesium silicate hydroxide.

Organic polymers as binders such as complex indigestible carbohydrates (cellulose, polysaccharides in the cell walls of yeast and bacteria e.g. glucomannans, and peptidoglycans, and others), and synthetic polymers such as cholesteryamine and polyvinylpyrrolidone can adsorb mycotoxins. Indigestible dietary fiber has adsorbance potential for mycotoxin. Certain bacteria, particularly strains of lactic acid bacteria, propionibacteria, and bifidobacteria, appear to have the capacity to bind mycotoxins, including aflatoxin and some *Fusarium*-produced mycotoxins. The binding appears to be physical with deoxynivalenol, diacetoxyscerpenol, nivalenol, and other mycotoxins associated with hydrophobic pockets on the bacterial surface. Other binders like synthetic water-soluble polymer, polyvinylpyrrolidone (PVP), has been investigated as a binder of mycotoxins however authors conclude that there is insufficient information is available to make any recommendations at this stage.

There is excellent potential for binders to help manage the mycotoxin problem and various materials can bind mycotoxins in feed and thus reduce toxic exposure to consuming animals. However no product currently meets all the characteristics for a desirable binder. A binder must be effective at sequestering the mycotoxin(s) of interest and should significantly prevent animal toxicity. There should not be detrimental effects on the animal and costs should render its use practical, profitable and usable in commercial feed manufacturing situations. Animal/product residues of mycotoxins should not increase and there should be no detrimental effects on the animal food product. Mycotoxins in feeds should not be masked such that feed contamination cannot be verified.

**Conclusion**

This article barely touches on the vast amount of data available in the literature. The reader is advised to refer to the Merck Veterinary manual for its table on mycotoxins and various other authors for further information. It is important for veterinary practitioners to be aware of the erosive effects that mycotoxins may have and their possible involvement in a number of unusual clinical manifestations.

**QUESTIONS:**

1. *Aspergillus flavus* produces the toxin  
   a. Aflatoxin B1  
   b. Aflatoxin B2  
   c. Aflatoxin G1  
   d. Aflatoxin G2  
   e. All of the above

2. Aflatoxin B1 is a potential human health hazard  
   a. From liver residues in marketed animals  
   b. Through milk  
   c. Through residues in meat  
   d. Through eating contaminated grain
e. Through dust in animal feed factories

3. *Aspergillus clavatus* is associated with the following symptoms
   a. High temperature and suppressed heart rate
   b. Muscle fasciculation of the cranial muscles
   c. Epileptiform convulsions and death
   d. Hypermetria or knuckling of hind legs and hyperesthesia
   e. Leukoencephalomalacia in horses

4. The trichothecene group of mycotoxins include
   a. Deoxynivalenol and T-2 toxin
   b. Aflatoxin M1 and aflatoxin AFB2
   c. *Diplodea maydis*
   d. Fumonisin B1
   e. None of the above

5. *Aspergillus* species mainly colonise important grain crops
   a. During growth and harvest
   b. During storage
   c. During milling and processing
   d. Only in drought years
   e. In the absence of pollenating insects

6. T-2 toxin produced by fusarium moulds is
   a. Nontoxic to beef cattle
   b. Associated only with feed refusal in dairy cows
   c. Associated with feed refusal, enteritis and abomasal ulcers in calves
   d. Associated with severe epileptiform convulsions followed by complete recovery
   e. Associated with front limb lameness

7. Zearalenone is a toxin produced by
   a. *Diplodea maydis*
   b. *Aspergillus fumigatus*
   c. *Penicillium spp*
   d. *Fusarium spp*
   e. *Aspergillus clavatus*

8. Identification of multiple toxins simultaneously can be achieved using
   a. Black lighting
   b. Mold spore counts and identification
   c. Liquid chromatography mass spectrometry
   d. PCR tests
   e. None of the above

9. Ideal mycotoxin binders used for in feed limitation of toxin damage in cattle
   a. Should sequester the mycotoxin and prevent signs of toxicity
   b. Should have no detrimental effects on the treated animal
   c. Should be cost effective
   d. Should not allow increase in mycotoxin residues in animal products
   e. All of the above

10. Strategies to prevent mycotoxin in crops should include
    a. Prevention of insect damage in crops
    b. Storing grain at higher humidity levels
c. Storing grain at low humidity levels
d. a. and c.
e. a. and b.

ANSWERS
1. e
2. b
3. d
4. a
5. a
6. c
7. d
8. c
9. e
10. d
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