MYCOTOXINS: HORSE HEALTH RISK?

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Catherine Dunnet cuts through the hype surrounding mycotoxins, and evaluates scientific evidence behind the problem, as well as management techniques

MYCOTOXINS are secondary fungal metabolites that, when ingested, inhaled or absorbed through the skin, can cause pathological changes in animals, contributing to reduced performance, sickness or even death in some instances.

As secondary metabolites, mycotoxins have no functional significance to the fungus itself, yet they represent a potential hazard to horse health.

Mycotoxin contamination has been reported in forages, cereals and other raw materials that are found routinely in horses' diets. While there are estimated to be more than 300 types of mycotoxins, *Fusarium* (Figure 1), *Penicillium* and *Aspergillus* are the most significant fungi with respect to equine health.

The most commonly reported mycotoxins associated with forage and other feedstuffs are described in Table 1.

Prevalence and relevance

The prevalence of mycotoxins in horse feed and forage has previously been shown to be significant. Data presented by Buckley (2007), revealed that 50 per cent of Irish hay, 37 per cent of haylage and 13 per cent of Canadian hay (sampled from Irish racing yards over a period of 12 months – 2002 to 2003), were contaminated with one or more of the following pathogenic fungi:

Aspergillus fumigatus, *A niger* or *A flavus oryzae*.

The mycotoxin zearalenone (ZEN) was present in 21 per cent of the Irish hay samples, but only eight per cent of the haylage and Canadian hay, respectively.

Samples of coarse mix, oats and pelleted feed revealed a lower level of mould and mycotoxin contamination, with the pelleted feed having the lowest content. Both *Aspergillus* and *Fusarium* mould species were isolated, with the mycotoxins ZEN, T2-toxin, fumonisin and ochratoxin being present (see Table 2). The relatively high mycotoxin level observed in the haylage should perhaps be viewed with caution, as no details were provided as to the quality of the haylage sampled, the degree of fermentation or the stability of the product.

With the exception of performance yards, forage tends to be purchased relatively locally; whereas cereals and ingredients for concentrate-feed manufacture are often sourced further afield. The worldwide occurrence of mycotoxins is, therefore, of great relevance to the equine industry.

A large-scale survey of mycotoxin contamination in feed ingredients, including maize, soya bean meal, wheat bran and rice bran, was carried out over two years in the European, Mediterranean and Asian-Pacific markets. Analysis of the 2,753 resultant samples for six *Fusarium* mycotoxins, as well as ochratoxin A and aflatoxin B, revealed that more than 50 per cent of the European and Mediterranean samples and 30 per cent of the Asian-Pacific samples were contaminated with one or more mycotoxins (Binder et al, 2007).

Samples from Europe were primarily contaminated with deoxynivalenol (DON), ZEN and T2-toxin, while those from the Asia-Pacific region tended to be contaminated with DON, ZEN, fumonisins and aflatoxin.

Mycotoxins can be formed in the field prior to harvest, or subsequently during storage or feed manufacture. The level and type of fungus, and the associated mycotoxins produced, are highly dependent on environmental conditions, particularly temperature and humidity, as well as the amount of moisture available in the host crop.

However, the relationship between the level of mould and the mycotoxins present in forage and feed is not straightforward. While a high total mould count in hay, for example, may harbour high mycotoxin content, the converse is not always true. A low mould count does not preclude a high mycotoxin concentration. Mycotoxin analysis in forage is warranted, therefore, as subjective assessment of hay is inherently unreliable (Raymond et al, 2000), and simple mould and yeast counts are unlikely to discount the presence of significant mycotoxin contamination.

Screening forage for mycotoxins can be carried out routinely by a number of methods, including enzyme-linked immunosorbent assay or liquid chromatography tandem mass spectrometry. However, while within economical reach of feed and forage manufacturers, and veterinary

practices representing large commercial yards, the costs mean routine use by vets for single-horse owners (Figure 2) and smaller trainers is limited.

Health hazard?

The potent effects of ingesting forage or feed that has a relatively high level of mycotoxin contamination in horses are well documented.

For example, stachybotryotoxicosis is one of the earliest recognised mycotoxin-mediated diseases. It was linked to the illnesses and death of thousands of horses in eastern Europe and Russia in the 1930s, with a further outbreak in Hungary in the mid-1980s (Newman, 2005). Furthermore, fescue toxicosis and ryegrass staggers, the symptoms of which are both mediated by mould and mycotoxin ingestion, are relatively well-known diseases in horses.

Fescue toxicosis is characterised by reproductive problems, such as increased gestation length, agalactia and mare and foal mortality (Cross et al, 1995), while ryegrass staggers involve neurological signs, such as lack of coordination, head shaking and collapse (Hunt et al, 1983 and Nollet et al, 2007). While the incidence of these overt mycotoxinderived diseases is considered rare, there is growing concern in both human and animal health circles regarding the chronic ingestion of low-grade mycotoxin contamination's effects on health. While the prevalence of mycotoxin contamination and the potential exposure to horses through feed and bedding cannot be disputed, the significance of this level of contamination and the clinical relevance of chronic low-grade intake largely remains to be established.

Mycotoxin intake in other species has been reported to contribute to a generalised failure to thrive, which was linked to reduced feed intake, feed refusal and poor feed conversion, as well as reduced reproductive capacity, immune suppression and an increased incidence of secondary infections (Fink-Gremmels et al, 2007 and Morgavi et al, 2007). In dogs, contamination of pet food with mycotoxins has historically led to many deaths, with 16 major mycotoxin outbreaks recorded between 1974 and 2006 (Leung et al, 2006).

The clinical picture in reported horses, however, is less clear, which may be related to the degree of feed contamination and the duration of exposure.

Johnson et al's (1997) study showed no appreciable effect of feeding barley contaminated with DON to non-pregnant mares and geldings in terms of feed intake, but circulating levels of IgG and IgA were reduced. However, these animals were at grass, so the barley only represented a relatively low proportion of the total feed intake, which would have greatly diluted the mycotoxin concentration in the total ration.

In contrast, horses fed a diet contaminated with 15mg/kg DON, in conjunction with other mycotoxins – specifically 15-acetyldeoxynivalenol (0.8mg/kg), fusaric acid (9.7mg/kg) and ZEN

(0.2mg/kg) – exhibited reduced concentrate feed intake compared to non-contaminated control feeds, despite a relatively lower intake of DON compared to the previous trial (Raymond et al, 2003). This may reflect a more intensified response to DON due to interaction between the different mycotoxins present that more accurately reflects natural contamination.

Leung et al (2006) discussed the possibility that exposure to mycotoxin cocktails can intensify the clinical response in some instances, compared with exposure to a single mycotoxin. Further experimental studies, or case reports, have also discussed clinical signs of toxicity to other mycotoxins, including aflatoxin, ZEN and fumonisins in horses, as reviewed by Newman (2005).

The aetiology of colic is obviously multifactorial, but epidemiological studies have cited various aspects of diet as significant risk factors (White, 2005; Archer et al, 2006). Mixed mycotoxin ingestion has previously been proposed as a further potential factor in the development of colic (Barnett et al, 1995).

The mycotoxin DON was found in the concentrate feed of 100 per cent of the colic cases studied, while T2-toxin and ZEN were found above an analytical threshold concentration in 31 per cent and 44 per cent of the colic cases, respectively.

The level of contamination with DON in the non-colic horses, however, was also relatively high (70 per cent), although neither T2-toxin nor ZEN were found above the analytical threshold in these control horses. This may suggest synergistic mycotoxin effects on the clinical outcome of ingestion.

However, while these results are suggestive, they are by no means unequivocal, as the sample size was very small (n=26) and only the concentrate samples were statistically analysed, as forage samples were not provided by all farms (Barnett et al, 1995). The practice of soaking reduced-quality hay, however, should perhaps be re-examined, as although it may reduce the exposure of horses to airborne moulds and mycotoxins, it may still represent a mycotoxin risk through ingestion.

The effects of mycotoxin ingestion are relatively well reported. However, a link has also been made between inhalation of mycotoxins and the development of recurrent airway obstruction in horses (Feige et al, 2002 and Larsson et al, 2003).

Management strategies

Unfortunately, mycotoxins are relatively resistant to chemical or thermal treatment, so once they are formed, they are a challenge to remove from feed ingredients or finished feed.

A number of strategies to reduce the mycotoxin load to animals have been investigated, ranging from pre-harvest treatments and management regimes to reduce the formation of mycotoxins, to storage strategies for cereals and the inclusion of ingredients with mycotoxinbinding properties, or enzymatic activities to reduce the in vivo absorption of mycotoxins.

• **Pre-harvest techniques**

These techniques involve the use of disease-resistant varieties to reduce mycotoxin formation. In addition, certain field management protocols, such as crop rotation and irrigation, have been reported to influence mycotoxin formation in the field (Kabak et al, 2006).

• **Processing techniques**

This is the use of sieving to remove cracked and damaged grains, as these are more likely to harbour fungal growth.

Washing grains can also reduce the mycotoxin load, as they are primarily found on the outer surface of the grain. Acid-based mould inhibitors are used extensively by the feed and forage industry, including the equine sector, to prevent mould and mycotoxin formation. Weak organic acids, such as acetic, sorbic and propionic acids, are known to inhibit fungal growth (Leung et al, 2006).

• **Nutrient supplementation**

A number of nutrients, such as polyunsaturated fatty acids, branched chain amino acids and antioxidants (including vitamin E and selenium), have also been proposed to minimise the tissue damage and altered behaviour that can be characteristic of mycotoxicosis. Organic selenium, in combination with a yeast-derived mycotoxin binder, has been evaluated to help protect the liver against lipid oxidant damage and antioxidant depletion, as a consequence of mycotoxin exposure in other species (Dvorska et al, 2007).

• **Bacterial deactivation**

Some micro-organisms, including certain *Lactobacillus* species, have been reported as having potential for detoxifying mycotoxins in animal feed and forage (Fuchs et al, 2008), although no work in this area has been carried out in horses.

• **Sequestering agents**

These ingredients have physical properties that facilitate the binding of mycotoxins within the gut, thus preventing – or at least reducing – their absorption by the animal. Numerous mycotoxin-binding agents are available commercially, including bentonites, aluminosilicates, zeolites and glucomannan derivatives.

The mycotoxin-binding agent choice is important, as many do not have sufficient binding activity for the wide range of mycotoxins that can potentially be present in feed or forage. Equally, a good mycotoxin binder must have the ability to retain mycotoxins in the bound state over the range in

physiological pH that occurs in the equine digestive tract, nor should they adversely affect nutrient intake by having unwanted binding effects on components of the diet. As far as horses are concerned, the only mycotoxin-binding ingredients that have been investigated consist of glucomannan-containing polymers derived from yeast cell walls. A mycotoxin binder derived from yeast (*Saccharomyces cerevisiae* 1026) was used to supplement concentrate feed that was experimentally contaminated with a range of *Fusarium* mycotoxins, as described earlier (Raymond et al, 2003).

The effect of the mycotoxin binder, which was fed at a level of two per cent (approximately 5g/day), was then compared to a control diet and also to the same mycotoxin-contaminated diet, without the addition of the mycotoxin binder. It was effective in alleviating some clinical indicators of mycotoxicosis (*Fusarium* species) observed when the unsupplemented mycotoxincontaminated diet was fed.

The mycotoxin-contaminated diet induced appetite suppression and increased serum gamma-glutamyltransferase activity, both of which were ameliorated by the addition of the yeast-derived mycotoxin binder (Raymond et al, 2003). However, these results were not repeated in a subsequent trial and the authors suggested that the beneficial effect was likely to relate to the mycotoxin contamination's extent and the degree of clinical signs (Raymond et al, 2005).

Summary

In summary, mycotoxin contamination of feed, forage and bedding is a reality for all horses, but may be particularly significant for horses undertaking athletic or breeding activities.

Here, the potential deleterious effects of mycotoxins on appetite, immune function, fertility and overall health and well-being may limit exercise or breeding performance. The presence of mycotoxin contamination in feed, forage and bedding is certainly worth investigation where continued poor performance is experienced, despite any investigated cause. While there is much to research into the clinical impact of mycotoxins in horses, it would appear prudent to take active steps to reduce horses' exposure to mycotoxins.

References

- Archer D C and Proudman C J (2006). Epidemiological clues to preventing colic, *Veterinary Journal* **172**(1): 29-39.
- Barnett D T, Mowery R A, Hagler W M and Bristol D G (1995). The correlation of selected mycotoxins to the incidence of colic in horses, *Proceedings of the 14th Equine Nutrition and Physiology Symposium*.
- Binder E M, Tan L M, Chin L J, Handl J and Richard J (2007). Worldwide occurrence of mycotoxins in commodities, feeds and feed ingredients, *Animal Feed Science and Technology* **137**(3-4): 265-282.

- Buckley T C (2007). Analysis of Canadian and Irish forage, oats and commercially available equine concentrate feed for pathogenic fungi and mycotoxins, *Irish Veterinary Journal* **60**(4): 231-234.
- Cross D L, Redmond L M and Strickland J R (1995). Equine fescue toxicosis: signs and solutions, *Journal of Animal Science* **73**(3): 899-908.
- Dvorska J E, Pappas A C, Karadas F, Speake B K and Surai P F (2007). Protective effect of modified glucomannans and organic selenium against antioxidant depletion in the chicken liver due to T2-toxin-contaminated feed consumption, *Comparative Biochemistry and Physiology – Toxicology and Pharmacology* **145**(4): 582-587.
- Fink-Gremmels J and Malekinejad H (2007). Clinical effects and biochemical mechanisms associated with exposure to the mycoestrogen zearalenone, *Animal Feed Science and Technology* **137**(3-4): 326-341.
- Fuchs S, Sontag G, Stidl R, Ehrlich V, Kundi M and Knasmuller S (2008). Detoxification of patulin and ochratoxin A, two abundant mycotoxins, by lactic acid bacteria, *Food and Chemical Toxicology* **46**(4): 1,398-1,407.
- Hunt L D, Blythe L and Holtan D W (1983). Ryegrass staggers in ponies fed processed ryegrass straw, *Journal of the American Veterinary Medical Association* **182**(3): 285-286.
- Johnson P J, Casteel S W and Messer N T (1997). Effect of feeding deoxynivalenol-(vomitoxin)-contaminated barley to horses, *Journal of Veterinary Diagnostic Investigation* **9**(2): 219-221.
- Kabak B, Dobson A D W and Var I (2006). Strategies to prevent mycotoxin contamination of food and animal feed: a review, *Critical Reviews in Food Science and Nutrition* **46**(8): 593-619.
- Leung M C K, Diaz-Llano G and Smith T K (2006). Mycotoxins in pet food: a review on worldwide prevalence and preventative strategies, *Journal of Agricultural and Food Chemistry* **54**(26): 9,623-9,635.
- Morgavi D P and Riley R T (2007). An historical overview of field disease outbreaks known or suspected to be caused by consumption of feeds contaminated with *Fusarium* toxins, *Animal Feed Science and Technology* **137**(3-4): 201-212.
- Newman K E (2005). Mycotoxins in equine diets: the difference between win, place and show. Nutritional biotechnology in the feed and food industry. *Proceedings of Alltech's 21st Annual Symposium*, Lexington, Kentucky, Nottingham University Press. Nollet H, Vanschandevijl K, Lefere L and Deprez P (2007). First confirmed case of ryegrass staggers in horses in Belgium, *Eerste Geval van Raaigraskramp bij Paarden in Belgie* **76**(5): 355-358.
- Raymond S L, Heiskanen M, Smith T K, Reiman M, Laitinen S and Clarke A F (2000). An investigation of the concentrations of selected *Fusarium* mycotoxins and the degree of mould contamination of field-dried hay, *Journal of Equine Veterinary Science* **20**(10): 616-621.
- Raymond S L, Smith T K and Swamy H V L N (2003). Effects of feeding a blend of grains naturally contaminated with *Fusarium* mycotoxins on feed intake, serum chemistry, and haematology of horses, and the efficacy of a polymeric glucomannan mycotoxin adsorbent,

Journal of Animal Science **81**(9): 2,123-2,130.
- Raymond S L, Smith T K and Swamy H V L N (2005). Effects of feeding a blend of grains naturally contaminated with *Fusarium* mycotoxins on feed intake, metabolism, and indices of athletic performance of exercised horses, *Journal of Animal Science* **83**(6): 1,267-1,273.
- White N A (2005). *Prevalence, Demographics, and Risk Factors for Colic Prognosis and Strategies for Prevention*. American Association of Equine Practitioners focus meeting on colic, Quebec.

Figure 1. Fusarium-contaminated wheat – a potential source of mycotoxins that are harmful to horses.

Photo courtesy of NIAB/CHRISTINE LONG.

Figure 2. Screening could prove to be expensive for single-horse owners.

Fungi class	Associated mycotoxins
Aspergillus	Aflatoxin B1 B2, G1, G2 Ochratoxin A Cyclopiazonic acid (CPA)
Claviceps	Ergot alkaloids Clavines Lysergic acid Lysergic acid amides Ergopeptines (ergotomine, ergovaline)
Fusarium	Fumonisin B1, B2 and B3 Trichothencenes T2-toxin, HT-2 toxin, diacetoxyscirpenol (type A) Nivalenol, deoxynivalenol, fusarenon-x (type B) Zearalenone
Penicillium	Citrinin, roquefortine, CPA, patulin
Neotyphodium	Tall fescue toxins Ergot alkaloids, lolines, peramine, lolitrems

Table 1. Significant fungi and related mycotoxins

	Number sampled	Percentage containing pathogenic fungi	Fungi isolated	Associated mycotoxin
Irish hay	62	50 per cent	*Aspergillus fumigatus* *A niger* *A flavus oryzae*	Zearalenone
Haylage	54	37 per cent	*A fumigatus* *A niger* *A flavus oryzae*	Zearalenone
Canadian hay	63	13 per cent	*A fumigatus* *A niger* *A flavus oryzae*	Zearalenone
Coarse mix	38	13 per cent	*A niger* *A flavus oryzae* *Fusarium*	Zearalenone Ochratoxin T2-toxin Fumonisin
Oats	26	Eight per cent	*A niger* *A flavus oryzae*	T2-toxin
Pelleted feed	51	Four per cent	*A flavus oryzae*	Zearalenone T2-toxin

Table 2. Survey of feed and forage samples in Ireland (Buckley 2007)