How efficient is rumen elimination of mycotoxins? – Part 1

by Dr Henk Ghesquiere, Impextraco NV, Wiekevorstesteenweg 38, B-2220 Heist-op-den-Berg, Belgium.

Cattle farmers are well aware that moulds or fungi are detrimental to their production results. For years, the precautions to avoid moulding of feedstuffs have been passed down from father to son. Modern science confirmed the necessity of such precautionary practices: from time to time, fungal cells from feedstuffs were shown to also grow in living animals. In this manner, the mechanisms were elucidated for mycotoxins, which are diseases caused by living fungi.

In 1960, the disastrous ‘Turkey X disease’ pushed society to also elucidate the mechanism behind it, which lead to the discovery of aflatoxin. This was the first recognition of mycotoxocoses, which are diseases caused by toxins that are produced by fungi.

In the 1970s and 1980s, it was revealed that rumen microflora may establish detoxification mechanisms for mycotoxins. Afterwards, attempts were made to transfer such detoxification mechanisms to seeds for crop production and to the gastrointestinal tract of monogastric animals, however with only limited success.

Within the cattle industry, such communications installed a false feeling of safety; therefore, it is appropriate to re-evaluate the efficiency of rumen mycotoxin elimination.

Transformation mechanism

The rumen microbial community is not only dense, but also diverse. The micro-organisms are known to digest the fibre fraction, which is indigestible for the host. Such microbial variety also facilitates transformation of other compounds, such as certain mycotoxins. Those effects vary from detoxification to amplification of toxicity.

Detoxification

- Ochratoxin A (OTA) is cleaved into phenylalanine and Ochratoxin-alpha (OTxα); since both reaction products are non-toxic, this is an effective detoxification.

There is an alternative pathway in which OTA is hydrolysed by a lactonohydrolase, but, since the end product is as toxic as the original OTA, this is not really helpful. Anyhow, the rumen microflora offers a certain level of protection against OTA. That is why the safety threshold is considered 7-10 times higher in cattle compared to swine.

Trichothecenes are a group of more than 180 compounds, which mostly share an epoxide moiety at position C12-C13. The rumen microflora may transform those mycotoxins by de-epoxidation into harmless metabolites. So, T-2 does not cause frequent problems in cattle, while it is one of the major concerns in monogastrics.

- Deoxynivalenol (DON) is in the same manner detoxified in the rumen, but inflammatory symptoms such as mastitis and laminitis were still observed in cattle. Since DON occurs in such a wide range of cattle feedstuffs (grains, stages and straw), an overload of the detoxification capacity is possible; when the rumen ingests more toxin than the microflora can cope with, unchanged DON still reaches the small intestine. Such overload is further intensified by other co-occurring trichothecenes, such as fusaric acid. So, detoxifying mechanisms allow cattle to withstand a four to five times higher contamination level of DON than pigs, but once this limit is surpassed, DON-mycotoxicosis does also occur in cattle.

Increased toxicity

- Zearalenone (ZEA) is produced by several Fusarium spp. In pigs, the mycotoxin is well known for its oestrogenic effects.

In the rumen, ZEA is readily transformed mainly to α-zearalenol and to a small fraction of β-zearalenol. Early literature considered the disappearance of ZEA as a detoxification, however α-zearalenol is three to four times more oestrogenic than the original ZEA.

While α-zearalenol is hardly absorbed from the gut, the subsequent metabolite is α-zearalanol or Zeranol. This Zeranol is readily absorbed and well known for its oestrogenic effects in cattle. So, there is no ZEA detoxification; the contrary is true.

For ZEA detoxification, a lactonohydrolase has been isolated; however such enzyme has not been found in the rumen or its microflora.

- Patulin is produced by Penicillium, Aspergillus and Bysschchlamus spp. Only in a limited number of feedstuffs, such as spoiled distillery and Continued on page 17
Continued from page 15
apple by-products, patulin contamination is high enough to cause the typical neurotoxic symptoms.

Nevertheless, in many silages, the patulin concentration reaches levels that are detrimental for the rumen microflora; such reduction of the microflora, reduces the detoxification capacity for OTA, T-2 and DON. Thus, patulin has a synergistic effect on other mycotoxins.

At this moment, some 300-500 different mycotoxins were identified; this figure is still on the rise. Some authors estimate the total number of mycotoxins at several thousands.

For most of those mycotoxins, there simply are no observations in animals; so evidently the rumen transformation and their effects in ruminants also remain unknown.

Different origins

● Grain related mycotoxins:

Once health authorities become aware that several mycotoxins show carcinogenic or teratogenic effects, more funding became available for mycotoxin research. Since both humans and animals consume grains, the focus for research has mostly been on grain mycotoxins.

Monogastrics consume similar grains as humans and more by-products; so, a lot of mycotoxin knowledge is interchangeable. In ruminant nutrition, such knowledge is also useful for evaluation of concentrates; however, in silages and pastures, many additional issues occur, that are unknown in human nutrition.

● Silage related mycotoxins:

When considering mycotoxins in silages in order to preserve the palatability as well as the nutritional value of the feeds. Such precautions are good practice against further development of moulds and mycotoxins.

● Traditional mycotoxins:

With respect to mycotoxins, storage is not the only concern. Most traditional mycotoxins are already produced when the crop is in the field and remain present in the silage. While mycotoxins are invariably invisible, the chopping of the crop spreads both the mould lesions and the mycotoxins, thus hiding the mould presence; chopping thus installs a false feeling of safety towards mycotoxin presence.

Nevertheless, the commonly known mycotoxins appear in similar levels in forages as they do in grains; Fig. 1 shows data from a large scale review (more than 8000 assays) performed over a period of nine years in North Carolina, USA.

This highlights that contamination of silages is as cumbersonse as in grains.

Special silage mycotoxins:

Due to the specific conditions (low oxygen and reduced pH), other moulds dominate in silages than in grains.

Therefore, apart from the field mycotoxins, silages often contain different storage mycotoxins. Examples of silage specific moulds are: Penicillium roqueforti (PR toxin, patulin, roquefortine, penicillic acid), Byssochlamys spp. (patulin, bysschlamic acid), Monascus ruber (monalins, citrinin).

Since the concerned mycotoxins are less frequently assayed than the typical grain mycotoxins, their influence remains largely under estimated.

A German investigation discovered moulds in 206 of 233 (88%) samples from grass or corn silage; dominant strains were Penicillium followed by Mucoraceae, Monascus and Aspergillus.

Mycophenolic acid, a mycotoxin that is hardly known in monogastric nutrition, was present in almost one third of the samples.

At the time of feeding silage:

Exposure to air is a contributing factor to mycotoxin development. At the time of feeding the silage, a certain level of exposure to oxygen is inevitable: at the surface from which the roughage is scraped off as well as in the feeding bunks.

The risk of mycotoxin production peaks under those circumstances. In corn silage, 25% of the samples from the core were positive for roquefortine C (average 96 µg/kg), while 0% of samples contained mycophenolic acid; for samples from the surface, those figures raised to 50% (average 1605 µg/kg) and 50% (average 660 µg/kg) respectively, while 100% of samples from moulded spots were positive for roquefortine C (average 25986 µg/kg) and 71% for mycophenolic acid (average 9311 µg/kg).

Such figures demonstrate the importance of so-called ‘hot spots’ in silages. Such investigations allow distinguishing field mycotoxins: almost all samples were positive for DON (average 950 µg/kg), regardless whether core, surface or moulded spots were analysed.

For ZEA, 50% of core samples (average 146 µg/kg), 44% of surface samples (average 137 µg/kg) and 29% of moulded spots samples (average 73 µg/kg) were positive. The much higher concentration of roquefortine and Mycophenolic acid in the moulded spots suggests those were produced during storage; DON and ZEA show the pattern for field mycotoxins that are evenly spread during chopping of the crop; the fermentation flora may have metabolised part of the ZEA.

Pasture mycotoxins

Although research on moulds and mycotoxins has been very limited in pastures when compared to grains, certain pasture diseases were that striking that elucidation of their cause was necessary.

Fescue toxicosis may cost more than five litres of milk or reduce growth rate by over 500g a day; some cattle may lose their hooves. The major grass in the US, tall fescue, is commonly infected by Neothypodium spp. and Claviceps spp., which produce a range of alkaloids of which ergovaline is measured as a marker toxin.

Ryegrass swards causes production losses, but mostly becomes evident only at the moment neurologic symptoms occur. In Europe, Oceania and South-America, rye grass (Lolium perenne) is the most common pasture grass.

Rye grass is commonly infected by Neothyphodium lolii that produces toxins of which lolitrem B is best known. Interestingly, a metabolite such as peramine protects the cold season grasses against insect attacks, which explains why the infection is so widespread.

Black patch disease or Slobbers disease is characterised by increased saliva production (often 50% more), while dry matter intake drastically goes down.

The fungus Rhizoctonia leguminicola causes black patches in legumes such as red clover; the mycotoxins, slaframine and swainsonine, produce neurologic symptoms such as the excess salivation (slobbers).

Apart from diseases that are exclusively linked to pastures, the other mycotoxins may also occur. In New Zealand and Australia, some 20-30% of grassland is known to contain ZEA levels above 1.0mg/kg DM, thus impairing fertility and reproduction of sheep and cattle.

A mix of mycotoxins

Obviously, there are many unknown factors when considering mycotoxins in ruminants. Should the farmers check grains, silages as well as pastures for mycotoxins? If so, which mycotoxins should be assayed?

Is there a transformation in the rumen and, if so, is it a detoxification or an amplification of the toxic effects?

Therefore, many farmers implement the best possible protection against mycotoxins in the TMR.

While a simple binder only offers some kind of protection against aflatoxin, a product eliminating a wider range of mycotoxins will further improve production performance.