How efficient is rumen elimination of mycotoxins? – Part 2

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In part 1 of this article (International Dairy Topics, Volume 12, Number 3) it was highlighted that the rumen may detoxify ochratoxin A, T-2 and DON, but can also amplify the effect of zearalenone. For most of the mycotoxins however, the effect of the rumen is still unknown.

For mycotoxins in grains, a lot of knowledge is available from human and monogastric nutrition; supplementary to those traditional mycotoxins, ruminants also cope with other types of mycotoxins in silages and pastures.

Safe levels of mycotoxins

When reviewing literature, there are remarkable differences in the recommendations for the levels of mycotoxins that are considered 'safe'. The maximum level of aflatoxin M1 that is tolerated in milk for human consumption, is one of the most striking differences: the FDA allows a level of 0.50ppb in the US, while a ten times lower limit is imposed in the EU: 0.05ppb!

Since opinions are that far apart for milk for human consumption, it is logical that even bigger differences are found in recommended levels for feedstuffs.

Fig. 1 shows the difference in recommended levels between cattle and pigs; pigs are considered the most sensitive animals, cattle the least susceptible animals.

Obviously, there are huge differences for certain mycotoxins; however, the link to the earlier described detoxification mechanisms is not clear. Ochratoxin and DON are detoxified in the rumen, so it is logical that there is a higher figure for cattle than for pigs.

But T-2 is also de-epoxidised in the rumen, while a similar level is recommended for both cattle and pigs. ZEA is transformed in the rumen to more oestrogenic products, but nevertheless the recommendation is some six times higher for cattle than for pigs. Lately, a lot of attention goes to contamination of feedstuffs by more than one mycotoxin. Since their effects are often additive or even synergistic, there is a tendency to lower the maximum limits even further. This is the case not only for ruminants, but also for monogastric animals.

Specific for ruminants is a wider variation in feedstuffs. Both silages and pastures are exclusively fed to ruminants. Since mycotoxins in roughages are less well investigated than those in grains, several effects still remain obscure, thus the recommended levels less reliable.

The meaning of figures

There is a huge variation in the recommended levels for ruminants. Certain authors focus their recommendation on the absence of clinical disease, while others focus on performance optimisation.

This partly explains the difference in figures: AFLA: 5-20ppb; DON: 50-2500ppb; ZEA: 70-3900ppb.

Maximum levels of aflatoxins in feeds and ingredients for dairy ruminants are defined at 20ppb by the FDA and at 5ppb only in the EU.

Macroeconomic observations are food for thought. The impact of human health authorities is on the rise. Whether farmers should perceive this as a burden or as a blessing is open for debate.

Prevention of subclinical mycotoxicoses improves milk yield, thus is also in the interest of the farmer. The calving interval is a lot higher in the US than in the EU; does stricter mycotoxin prevention contribute to a shorter calving interval?

If so, a reduction of one to two months (for example from 14 to 12 months), is financially highly beneficial.

Mycotoxins are linked to a diverse range of issues: mastitis, laminitis, abomasal displacement.

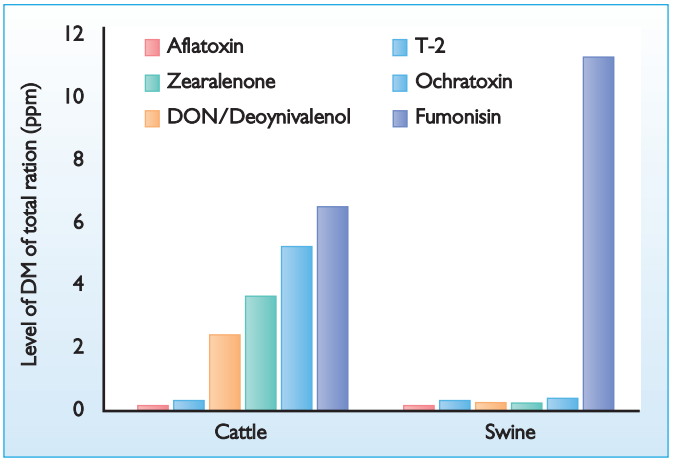
The current stage of knowledge is insufficient to calculate the cost/benefit ratio for each individual cattle farm; in practice, farmers often observe benefits from mycotoxin strategies that previously were not perceived as a problem. So, why not invest a bit to give it a try?

As for the variation in recommended limits and as mycotoxins are toxic products, there is a simple guideline: the lower, the better!

Dilution of contaminated products may avoid acute problems, but extends the duration of contamination. There is no way to eliminate mycotoxins from contaminated feedstuffs, what poses a dilemma: feeding the mycotoxin containing products to cattle or pay for their destruction?

In the end, the farmer must decide about continuing the use of contaminated feedstuffs or not.

Fig. 1. Comparison of recommended maximum limits of mycotoxins in cattle versus swine.



Detoxification

For detoxification of mycotoxins, different authors consider protozoa as the most important part of the rumen flora. Most protozoa do not survive outside and remain vulnerable even inside the animal.

That is why their identity and quantity fluctuate over time as a consequence of differences in diet or other variables. So, strains of detoxifying microflora may be present in one herd, but absent in the neighbouring herd. Detoxification is not a constant process.

Microbial strains that were identified with detoxifying activity are strictly anaerobic; consequently, those are hardly applicable in an aerobic environment such as compound feed.

As is the case with probiotics, it is not evident to transfer a bacterial strain from one animal species to another.

What is more, authors differ in opinion. As an example: some people consider Saccharomyces telluris as a detoxifying organism, while others claim this has not been proven.

Most research performed on such microbes focuses on isolating the genetic material encoding for the detoxifying enzymes; once this is done, this genetic material is transferred to enzyme producing strains with the goal to supplement feed with the concerned enzymes or transferred to crops in order to make such crops more mycotoxin resistant.

Mycotoxin detoxifying organisms are not only present in ruminants, but also in monogastrics. In one study, as much as 10 different detoxifying strains were isolated from the chicken gut.

Transformation of DON to the less toxic DOM-1 was observed with four different groups of bacteria (Clostridiales, Anaerofilum,

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Colinsella and Bacillus) isolated from poultry; such transformation is far more pronounced in the large intestine, than in the small intestine and only marginal in the crop.

Also in chickens, variation is at stake: some chickens harbour DON detoxifying organisms, others do not. Should this frighten chicken farmers? Not really; chicken are less susceptible to DON than pigs, mostly because of poor absorption from the chicken gut, while in pigs DON is already absorbed in the proximal small intestine; thus the effect of the microflora is limited in monogastrics.

Influences on the rumen

As previously described, the rumen detoxifies OTA, T-2 and DON, while it amplifies ZEA toxicity; for most mycotoxins, transformation has not yet been described.

What is more, the rumen flora highly varies from herd to herd, thus also the detoxification capacity. Detoxifying strains may disappear from a herd. SARA or subacute rumen acidosis is quite common in high producing dairy farms; higher levels of concentrates in the diet predispose to SARA.

Since protozoa are very sensitive to decline in pH, the mycotoxin degrading capacity of the rumen also disappears with a fall in pH. The simple introduction in the diet of readily fermentable sugars may eliminate the mycotoxin transforming capacity; once pH is normalised, this capacity may return or may remain absent during a prolonged period of time. Aflatoxin detoxification is very dependent on rumen pH, thus is hardly reliable.

Patulin is a mycotoxin that is known for its antimicrobial effect. Patulin may logarithmically reduce the rumen microflora. Therefore, its co-presence negatively influences mycotoxin detoxification.

Several mycotoxins are known for their immunity reducing effects. They contribute to the incidence of certain pathogenic bacteria; a link with a.o. Salmonella was shown. But, vice versa, pathogens cause gut damage which increases mycotoxin absorption. Since immune reduction is an aid in mould growth, it facilitates mycoses that further disturb the microbial balance in the rumen.

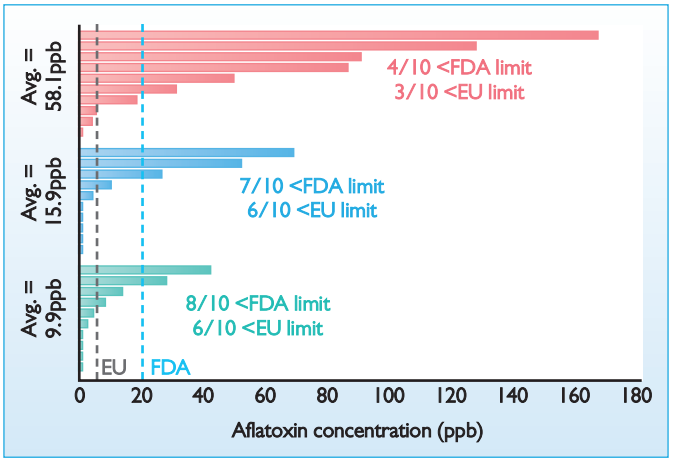

Fig. 2. How many samples should be assayed to accurately confirm a mycotoxin contamination?

Which approach?

● **Mould prevention:**
Evidently, prevention of moulding is important both in the field and during storage. Unfortunately, mycotoxins are invisible; while moulds can be present without mycotoxins, the inverse is also possible: feedstuffs free of signs of moulding may be highly contaminated by mycotoxins.

● **Mycotoxin assays:**
Assaying feedstuffs provides information about specific mycotoxins, but assays are expensive. Commercial laboratories routinely assay five to seven mycotoxins, which are commonly present in monogastric feed; thus, several important cattle mycotoxins often fall outside this scope.

Sampling procedures are not evident. As highlighted in part 1 of this article, many silage mycotoxins are concentrated in so-called 'hot spots'. A sufficient number of samples must be assayed before some accuracy is obtained. Fig. 2 shows this effect: for three different lots of corn silage, 10 samples of each were assayed for aflatoxin concentration.

For all three lots, the average contamination was above the EU limit (5ppb) for dairy feed, while only the average for the heavily contaminated lot was above the FDA limit of 20 ppb. Although heavily contaminated, 3-4 out of 10 samples were mistakenly considered as safe or even 'false negative'.

With a limited number of assays, missing or underestimating aflatoxin contamination is a considerable risk; also overestimation is possible, albeit the latter will not result in aflatoxin positive milk deliveries.

● **Feedstuff withdrawal:**
Assays are reliable for large scale statistics or epidemiologic studies. For each individual farmer, it is very elaborate and expensive to assay a wide range of mycotoxins in each feedstuff.

When suspecting a feedstuff, the common advice is to quit feeding the concerned feedstuff; symptoms disappearing or performance improvement after withdrawal is then proof for contamination. For further characterisation, assays are used. What to do with contaminated feedstuffs? Destruction is legally imposed in many countries, but is very expensive.

● **Mycotoxin elimination:**
Elitox is a combination product of enzymes, biopolymers, minerals, natural extracts and vitamin C, thus protecting against the widest possible range of mycotoxins.

Many cattle farmers have experience in using Elitox as a tool for elucidating the problem. Simple supplementation on top of the normal feed, often gives immediate effects that differentiate mycotoxin problems from other possible causes.

With ryegrass staggers, the Elitox effect is that spectacular that diagnosis often is feasible within 24 hours. During prolonged implementation, the farmer may consider a gradual replacement of the contaminated ryegrass parcels.

Also with TMR in which one or more feedstuffs are suspected, improvements are often observed within days: such as increased feed intake, dung consistency (less diarrhoea, blood or mucus) and alertness.

Once confirmed, it is only a matter of time before performance improvements become evident: higher milk yield, lower SSC, better fertility, etc.

Conclusion

Talks about rumen detoxification established a somewhat false feeling of safety; effects of mycotoxins are often underestimated in cattle.

Diversity in feedstuffs and inaccuracy of data or assay procedures for (silage and pasture) mycotoxins obscure the decision about discarding certain feedstuffs or not.

An additive that produces a clear distinction between symptoms and performance before and after its implementation has proven to be an effective and practical tool for dealing with mycotoxins in ruminant nutrition. ■