Optimising productivity and health in dairy cows

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Optimising productivity and health in dairy cattle, as anyone involved in dairy production knows, is a lot easier to talk about than to achieve.

The challenges that cattle confront daily are many and diverse from weather to pathogens to nutrition to housing to milking equipment and more. How well management handles these challenges is the key determinant in achieving optimum productivity and health in the herd. Too often the effects of mycotoxins on dairy animals are either unrecognised or misdiagnosed thus not properly managed. The objective of this article is to discuss mycotoxins in dairy rations and measures that may be taken by management to reduce the risk they pose to achieving and maintaining optimum productivity and health.

Feedstuff contamination

Mycotoxins can contaminate almost any feedstuff used in rations fed to animals, including dairy cattle. The mycotoxins come in feedstuffs that are infested by moulds that either have produced or may produce the toxin when conditions are right. Moulds which produce mycotoxins while crops are growing in the field (fusarium and aspergillus moulds) are referred to as 'field' moulds. 'Storage' moulds produce mycotoxins (penicillium and aspergillus moulds) in feedstuffs stored, usually under improper conditions, for future use. Sometime ago FAO estimated that 25% of the world grain production is contaminated with mycotoxins. It is possible that that number is somewhat conservative today. Further, estimates from Pioneer Hybrids are that some 95% of the mycotoxins present in feedstuffs are in the material at harvest. While only a small percentage may be formed during storage they are no less significant in posing challenges to dairy cattle. Mycotoxins from any source jeopardise animal health and performance.

Mycotoxin	Binding	Deactivation
Aflatoxins	+++	
Ochratoxins	+ –	+++
Fumonisins	+ –	+++
Zearalenone*	+ – –	+++
DON (vomitoxin)		+++
Nivalenol		+++
T-2 toxin		+++
DAS, MAS		+++
Other trichothecenes (3-AcDON, 14-AcDON, Fus X, HT-2 toxin, etc)		+++

* Some zearalenone may be bound but most must be deactivated enzymatically.

Table 1. Managing mycotoxins with a binder or an enzyme deactivator.

A number of factors influence the incidence of mycotoxins in feedstuffs. The greatest contributor is weather, and different moulds favour different weather conditions to flourish and produce mycotoxins.

Cultural practices such as low-till and no-till cultivation, and reduced crop rotation favour the overwintering of more mould spores, which results in a greater mould infestation of plants, and significantly contributes to incidence of mycotoxins.

In some years crops pose a greater risk from mycotoxins than others because weather plays such a major role.

The 2009 US corn crop is a good example as high levels of aflatoxins (aspergillus mould toxin) are found in the hot, drought affected southern states while deoxynivalenol (called DON or vomitoxin), fumonisins, and zearalenone (fusarium mould toxins) are being reported in grains across the breadth of the mid-west where the summer was cooler and damper than normal.

Similarly, Canadian grain crops are also reported to be contaminated with high levels of fusarium mycotoxins. Long term, the incidence of mycotoxins may increase due to greater adoption of minimum tillage practices; however, GM crops, which reduce insect damage, have been shown to have a lower incidence of mycotoxin contamination even though the moulds are present.

Other measures, such as introducing atoxigenic strains of aspergillus that compete with toxigenic strains, and selecting varieties of corn and other grains for resistance to toxigenic moulds hold promise to reduce the incidence of mycotoxins in feedstuffs.

Identifying mycotoxicoses

Today, the fact is that feedstuffs being fed to growing and lactating dairy cattle are contaminated with mycotoxins and some at very harmful levels. It would be great if one could easily identify a mycotoxicosis (disease process caused by mycotoxins).

Unfortunately, unless the mycotoxicosis causes dramatic changes such as a precipitous drop in milk production, lower feed intake or even sudden death it usually is difficult to know whether mycotoxins are the culprit or not. Feedstuffs can be analysed for mycotoxins; but one should not be too comforted by a laboratory report showing that no mycotoxins were detected.

Mycotoxins are not uniformly distributed in feedstuffs and getting a sample from contaminated areas, called hot spots, may not happen; thus, the sample sent for analysis does not accurately represent the feedstuff. Good sampling technique is essential but it does not guarantee that the results really represent the material. Also, it is possible that mycotoxins may not be detected in the laboratory because of side chains on the

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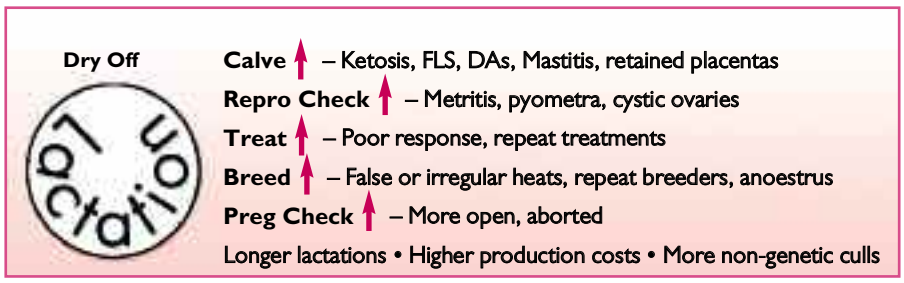
Fig. 1. Effects of subclinical ingestion of mycotoxins.



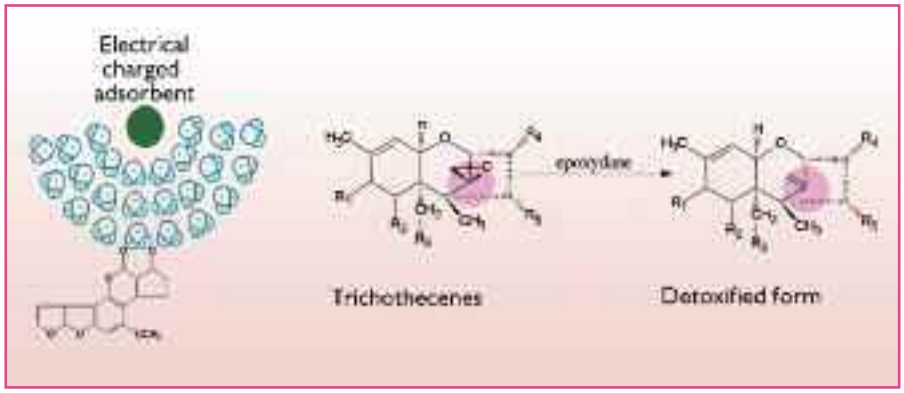


Fig. 2. Aflatoxin is a polar mycotoxin with sites located for easy binding.

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toxin which mask its detection but do not lessen its toxicity when consumed.

One can take heart with a positive analysis in that it allows decisions to be made on how to use the contaminated feedstuff. A luxury is being able to completely eliminate the contaminated feedstuff from the ration.

Such luxury is rare but rations may be adjusted such that less of the contaminated feedstuff is consumed daily and the likelihood of adverse effects can be reduced.

More often than not, feedstuffs are not analysed so it is not known whether or not impaired animal performance and health is caused by mycotoxins in the ration or some other cause.

As noted earlier, unless the effect of mycotoxins is severe they are not likely to be suspected and properly managed. Far more commonly, the ingestion of low levels of mycotoxins causes subclinical effects, which is a challenge to properly diagnose.

Subclinical effects

These subclinical effects appear as subtle increases in what may be considered common cow problems, especially in postpartum cows (Fig. 1).

It is well acknowledged that mycotoxins suppress the immune system, even at levels that may not cause metabolic or physiological problems.

Consequently, an increase in the number of post partum cows with retained placentas, mastitis or high SCC, metritis or poor response to antibiotic therapy, may be attributed to causes other than to feeding rations with low levels of mycotoxins.

Such contamination my also result in lowered DMI (dry matter intake), lower milk production and depressed butterfat. If the mycotoxin is zearalenone, the reproductive system can be affected and reproductive efficiency diminished.

Growing cattle may have slightly lower daily gain and not reach development goals; they may fail to develop the full immunity that they should when vaccinated, or they may experience premature sexual development. These subclinical effects of mycotoxins tend to lead to higher veterinary expense, higher production costs, longer lactations, and increased non-genetic culling, just to mention only a few of the decreases in productivity and health.

The challenge for management is to differentiate a mycotoxicosis from a veterinary problem, a nutritional problem or a problem created by management. A practical example of the difficulty is differentiating between a mycotoxicosis and a poor DCAD dry cow program that results in a subclinical hypocalcemia. The cows in each situation can experience a higher incidence of retained placenta, a bit more mastitis and feed intake and milk production can be less than it should be. As mentioned earlier, optimising productivity and health is easier said than done.

Going back to the various mycotoxins, for the purpose of discussing products that are useful in managing the risk of mycotoxins, the mycotoxins should be divided into two groups: those that are bindable (adsorbable) and those that are not bindable (not adsorbable). In turn, products that are available to help manage mycotoxins in feeds can also be divided into two groups: mycotoxin binders (adsorbents) and mycotoxin deactivators (products containing specific enzymes). Mycotoxin binders are composed of either various clay products or yeast cell wall products or the two can be combined. Mycotoxin deactivators are products which contain specific enzymes that act on certain groups in the various mycotoxins.

Table 1 shows the mycotoxins that can be bound and those which must be managed with a mycotoxin deactivator. Some can be managed with an appropriate binder or with a deactivator product.

Polar mycotoxins

Many mycotoxins are polar, but unless the polar site is suitably located the toxin cannot be adsorbed by a binder. Aflatoxin is a polar mycotoxin with sites located for easy binding (Fig. 2); thus, it can be managed with a number of good binders.

For the dairy industry this is important because of the penalty for producing milk with more M1, the aflatoxin metabolite secreted into milk when too much is fed, than is permitted by law. Not all binders are equal in their ability to bind mycotoxins, even aflatoxins; therefore, it is important to do the homework necessary to select a good binder. Price is not always an indicator of quality and should not be the sole criteria for selection. Fig. 2 also illustrates how enzymes render non-bindable mycotoxins non-toxic.

Trichothecenes, which are not bindable, have a ring structure (12, 13 epoxide ring) that makes them toxic. In the example, the epoxide ring in DON is altered enzymatically to the non-toxic deepoxide metabolite. Similarly, other enzyme can remove the menace of zearalenone, ochratoxins and fumonisins.

Identification difficult

Ideally, the kinds of mycotoxins present in feedstuffs would be known so that it could be managed more effectively either by limiting its use in rations or by selecting the most appropriate product to manage them.

In reality this is seldom the case, yet something must be done to maintain productivity and health. A part of doing the homework is to find out what kinds of mycotoxins are most likely to be in the feedstuffs being used in the rations. Aflatoxins are more likely to be found in corn coming from hot, dry areas, whereas DON, zearalenone and other fusarium mycotoxins predominant in cooler climates.

Today, the feedstuffs going into the ration are often not of local origin and little is known about which, if any, mycotoxins may be present. In such a situation it may be wise to choose a product that covers the spectrum by containing a binder and a deactivator.

Whatever the choice, the cattle should be observed closely. A persistent problem may indicate that either more of the product should be fed to accomplish the task, or it may mean that mycotoxins are not the problem. Identifying a mycotoxin problem, in most instances, is a process of elimination and feeding a mycotoxin product for 4-6 weeks is one way to narrow the list of possible causes of the problem.

Conclusion

Mycotoxins are, and will continue to be, a challenge to optimising performance and health in dairy herds for some time.

However, there are more tools available today to help managers minimise their impact on the herd. A few years ago the only feed additives available to manage mycotoxins were binders.

Today, managers can select a binder, if that is all that is needed, or they can select a product containing a mycotoxin deactivator and a binder to ensure that whatever mycotoxin is in the ration it will be managed properly. ■